South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 29 December 1956 Weekly 2s. 6d. | Vol. 30 No. 52 | Kaapstad, 29 Desember 1956 Weekliks 2s. 6d.

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Nuwejaarsgroete
van die
President

Dr. J. S. du Toit, President van die Mediese Vereniging van Suid-Afrika stuur die volgende boodskap van Nuwejaarsgroete aan lede van die Vereniging:

Dat die jaar 1956 na sy einde haas word weerspieël in alles om ons heen, ook in wat ons onderneem om nog te doen.

Laat ons probeer behou uit die afgelope jaar alles wat goed is; ook wat ons van waarde ag in die goeie gesindheid onder mekaar.

Met die koms van die jaar 1957 is dit my begeerte om vir al die lede van die Mediese Vereniging van Suid-Afrika 'n jaar van Vrede en Vreugde toe te wens.

Ek dink veral aan die ongeveer 280 jong lede wat hierdie jaar hulle loopbaan in ons eerbare roeping begin. Mag hulle, soos ons wat al baie jare in hegte eenheid in diens saamgesnoer is, ondervind dat die grootste beloning lê in die dankbaarheid van die mensdom wat deur ons gedien word.

New Year Greetings
from the
President

Dr. J. S. du Toit, President of the Medical Association of South Africa, sends the following message of greeting for the New Year to members of the Association:

That the year 1956 is fast drawing to an end is reflected in everything around us; as well as in what still remains for us to do.

Let us try and retain from the past year everything that is good, and that which we value in our friendly relations with one another.

With the coming of the year 1957, it is my desire to wish all the members of the Medical Association of South Africa a year of Peace and Joy.

About 280 young members this year start their career in our honourable calling, and my thoughts go out to them. May they, like us who have been associated for these many years in close unity of service, find that the greatest return for service lies in the gratitude of those whom we serve.

BOODSKAP VAN DR. A. W. S. SICHEL

Voorsitter van die Federale Raad en die Hoofkantoor- en Tydskrifkomitee

Nog 'n jaar het gekom en gegaan en die tyd is geskik om die waardering van die Mediese Vereniging van Suid-Afrika uit te spreek teenoor al die lede wat gedurende die afgelope jaar op die Raad en die Sentrale Komitees van die Vereniging self, en op al die Rade en Komitees van die Takke, Afdelings en Groepe binne die moedervereniging gedien het.

Nie almal besef die harde werk en die opoffering van tyd en geleentheid wat so baie van ons kollegas vryelik en met entoesiasme gee nie. Hulle arbeid het die masjinerie aan die gang gehou, alhoewel dit soms nie so vlot of so gou was soos ons dit graag sou wou gehad het nie. Ons is baie dankbaar en ons sê ‚dankie'.

Dit is ook passend dat ons ons dank en waardering uitspreek teenoor almal wat in die voltydse diens van die Vereniging is. Ons wil graag ons Redakteur, ons Sekretarisse en die hoofde van al ons Departemente met hulle staflede dank vir hulle lojale en getroue toewyding aan plig en vir hulle samewerking.

Mag die komende jaar een van vooruitgang en tevredenheid wees, ondanks die menige probleme waar teenoor ons te staan mag kom. Waar ek my vriende en medelede alle voorspoed en geluk toewens, weet ek dat ons op die lojaliteit en samewerking van u almal kan staat maak, sodat ons steeds die eer en waardigheid van die Mediese Professie kan handhaaf en ons Vereniging tot 'n puik organisasie opbou.

MESSAGE FROM DR. A. W. S. SICHEL

Chairman of Federal Council and the Head Office and Journal Committee

Another year has come and gone and the time is appropriate to express the appreciation and thanks of the Medical Association of South Africa to all members who have served during the past year on the Council and the Central Committees of the Association itself and on all the Councils and Committees of the Branches, Divisions and Groups within the parent body.

Not everyone realizes the hard work and the sacrifice of time and opportunity which so many of our colleagues give freely and with enthusiasm. Their labours have kept the machinery moving, though on occasion not as smoothly or as rapidly as it would have been our desire. We are very grateful and we say, 'Thank you'.

It is also fitting that we express our thanks and appreciation to all those who are in the full-time employ of the Association. We desire to thank our Editor, our Secretaries and the heads of all our Departments, with their staffs, for their loyal and faithful devotion to duty and for their cooperation.

May the coming year be one of progress and satisfaction in the face of the many problems which confront us. In wishing my friends and fellow-members good luck and happiness, I know that we can rely on the loyalty and cooperation of one and all in continuing to uphold the honour and dignity of the Medical Profession and in our endeavours to make the Association a worthwhile organization.

VAN DIE REDAKSIE : EDITORIAL

LEWERKOMA

'n Redelike benadering tot siektes van die lewer vereis aanhoudende hersiening. Op geen ander orgaan in die liggaam het moderne mediese navorsing so 'n gekonsentreerde en volgehoue aanval gemaak nie. En terwyl ontsaglike boekdele van wetenskaplike gegewens verskyn het, is noue korrelasie met die goedgevestigde en dikwels-aangetrefde kliniese patrone van lewersiekte nooit regtig bereik nie. Dit is nêrens so opvallend soos by die behandeling van hierdie toestande nie, waar dit skyn of die mees spesifieke geneesmiddel waaroor die dokter beskik, nog steeds bed-rus is; op weinig anders kan daar onomwonde aanspraak gemaak word dat dit net so doeltreffend is.

Simptome van geestesgesteldheid en koma is lank reeds as ernstige komplikasies—dikwels laat en teen die einde—van chroniese lewersiekte beskou. Die presiese meganisme van hulle voortbrenging het duister gebly en hul behandeling, simptomaties en grootliks proefondervindelik. Hierdie sogenaamde neuropsigiatriese sindroom is 'n komplikasie van verskillende tipes van lewersiekte en dit is verklaar dat sy voorkoms te wyte is aan die produksie van 'n toksiese stikstofhoudende

HEPATIC COMA

A rational approach to diseases of the liver requires constant revision. Upon no other organ in the body has modern medical research made so concentrated and sustained an attack. And whilst vast volumes of scientific data have appeared, close correlation with the well-established and oft-encountered clinical patterns of liver disease has never really been achieved. This is nowhere so apparent as in the treatment of these conditions, where the most specific remedy in the hands of the physician seems still to be bed rest; little else can be claimed unequivocally to be as efficacious.

Mental symptoms and coma have long been regarded as serious complications—frequently late and terminal—of chronic liver disease. The precise mechanism of their production has remained obscure and their treatment symptomatic and largely empirical. This so-called neuropsychiatric syndrome is a complication of diverse types of liver disease, and its occurrence has been explained as due to the production of a toxic nitro-

stof in die ingewande, wat deur die sieklike lewer in die sirkulasie van die liggaam en in die brein, ingebring word.[1] Voorts is dit aangeneem dat hierdie stof ammonia is, aangesien die bloed-ammonia waardes somtyds verhoog word.[2] Die teorie word gestaaf deur die feit dat lewerkoma by pasiënte met lewersiekte teweeggebring kan word der hulle met 'n dieet ryk aan proteïen of stikstofhoudende stowwe, soos byvoorbeeld ammoniumsoute of ureum, te voed.[3] 'n Grootse bydrae tot die begrip van hierdie belangrike onderwerp, is die onlangse studie deur Sherlock en haar kollegas by die Nagraadse Mediese Skool te Hammersmith, Londen, en die nuwe behoudende terapeutiese kursus wat vir die neuropsigiatriese sindroom aanbeveel is.[4] Die perke van die konvensionele betekenis van die term ‚lewerkoma' is aansienlik wyer gemaak, sodat die neuropsigiatriese sindroom enige kleiner variasie van bewussyn en neurologiese tekens, so wel as onopwekbare gevalle van koma, insluit. Tipies wisselend en hervattend—tot so 'n mate dat ‚herstel' sinoniem is met ‚geskik om huistoe gestuur te word'—word die sindroom omskryf as ‚die progressiewe belemmering van emosionele beheer en intellek, terwyl die pasiënt van bewustelooshеid na koma gaan.' Die klassieke ontlastingsruik, foetor hepaticus, is gedurig aan die asem van gevalle van dreigende koma te bespeur.[5]

Die Hammersmith reeks bestaan uit 66 opeenvolgende gevalle van lewersiekte wat deur ‚lewerkoma' bemoeilik was—dit is met laboratorium-, histologiese en lykskouingshulp oortuigend bewys dat almal sieklike lewers gehad het wat vir die kliniese eienskappe verantwoordelik was. Van hierdie 66 gevalle was 13, pasiënte met chroniese virus-lewerontsteking, 34 met akute lewersirrose, 13 met chroniese lewersirrose, en die oorblywende 6 het uit 'n kliniese mengsel (karsinoom, lewernekrose, ens.) bestaan.

Alle gevalle was behandel asof die ingewandstoksienteorie geldig was, en die volgende terapeutiese program is ingestel. Eerstens is die ingewande van stikstofbevattende stof deur lawemente gesuiwer (wat, so word dit gesê, die pasiënte geriefliker laat voel het), en dan deur die uitsluiting van proteïen uit die dieet, vry gehou. Koolhidraatopname is deur binnemaagse drup (20% glukose) en binneaarse politienbuise (40%) geforseer tot in die koparmaar om trombose in die kleiner are te vermy. Tydelike uitputting van stikstofhoudende proteïen is waarskynlik onskadelik by pasiënte met lewersiekte,[6] en by ‚lewerkoma'—maar slegs by koma—is dit die aangewese behandeling. Later, soos die pasiënt herstel, word klein hoeveelhede proteïen in die dieet ingesluit. Tweedens word chloortetrasiklien (Oureomisien) in volledige terapeutiese dossise toegedien. Eksperimenteel verhoed hierdie antibiotika massiewe lewernekrose by rotte,[7] en dit werk waarskynlik deur sy uitwerking op die organismes van die ingewande. Derdens word faktore wat bekend is om lewerskade te bespoedig—kalmeermiddels ammoniumsoute, ureum, verdowingsmiddels—vermy, en maagdermbloeding, veral van spatare van die slukderm, word deeglik behandel. Nege van Sherlock se 34 gevalle van akute lewersirrose was deur maagdermbloeding bespoedig; samepersing van die slukderm by wyse van 'n ingeslukte buis mag lewensreddend wees.

genous material in the intestines which is introduced through the disordered liver into the systemic circulation and the brain.[1] This toxic substance has further been held to be ammonia, since the blood-ammonia values are sometimes raised.[2] The theory is supported by the fact that hepatic coma can be induced in some patients with liver disease by feeding with a high-protein diet or nitrogenous substances such as ammonium salts or urea.[3] A major contribution to the understanding of this important subject is the recent study by Sherlock and her colleagues at the Postgraduate Medical School at Hammersmith, London, and the novel conservative course of therapy advocated for the neuropsychiatric syndrome.[4] The confines of the conventional meaning of the term 'hepatic coma' are considerably widened, so that the neuropsychiatric syndrome includes any minor variation of consciousness and neurological signs, as well as unrousable cases of coma. Typically fluctuant and recurrent—so much so that 'recovery' is synonymous with 'fit to be discharged home'—the syndrome is defined as 'the progressive impairment of emotional control and intellect, the patient passing from stupor into coma'. The classical faecal smell, foetor hepaticus, is constantly on the breath of cases of impending coma.[5]

The Hammersmith series comprises 66 consecutive cases of liver disease complicated by 'hepatic coma'—all conclusively proved by laboratory, histological or post-mortem aids to possess diseased livers responsible for the clinical features. Of these 66 cases, 13 were patients with acute viral hepatitis, 34 were acute biliary cirrhotics, 13 were chronic biliary cirrhotics, and the remaining 6 comprised a clinical miscellany (carcinoma, liver necrosis, etc.).

All cases were treated as if the intestinal-toxin theory was valid, and the following therapeutic programme was instituted. First, the intestine was cleared of nitrogen-containing material by enemata (which, it is said, made the patients feel more comfortable) and then kept free by the omission of protein from the diet. Carbohydrate was 'pushed' by intragastric drip (20% glucose) and intravenous polythene tubing (40% glucose) into the innominate vein to avoid thrombosis in the smaller veins. Temporary depletion of nitrogenous protein is apparently not harmful in patients with liver disease,[6] and in 'hepatic coma'—but only in coma—it is the treatment indicated. Later on, as the patient recovers, small amounts of protein are included in the diet. Secondly, chlortetracycline (Aureomycin) is administered in full therapeutic doses. Experimentally this antibiotic prevents massive hepatic necrosis in rats,[7] and it probably acts by its effect on the flora of the intestine. Thirdly, factors known to precipitate liver damage—sedatives, ammonium salts, urea, anaesthetics—are avoided, and gastro-intestinal haemorrhage, especially from oesophegeal varices, is vigorously treated. Nine of Sherlock's 34 cases of acute hepatic cirrhosis were precipitated by gastro-intestinal hae-

Die Hammersmith resultate is beslis beter as dié wat by enige vorige reeks van gevalle van lewerkoma verkry is. Van die 39 gevalle (58%) wat herstel het, was 21 in diep koma; in die besonder, het 6 van die 13 gevalle van virus-lewerontsteking herstel. Die reekse wat voorheen gerapporteer is, toon aansienlik hoër sterftesyfers (die aantal gevalle wat herstel het, soos deur Sherlock aangegee, word tussen hakies gewys): Foulk *et al.*, lewersirrose 52 (5); Katz en Ducci, virus-lewerontsteking 1,000 (*nul*); Stokes *et al.*, virus-lewerontsteking 23 (2); McDermott *et al.*, poortaarsirrose 20 (4). Die hoër sterftesyfers wat hier opgeteken is, mag te wyte wees aan verwarring oor die definisie van ‚lewerkoma', wat hierdie navorsers miskien tot die beskrywing van bewustelose pasiënte beperk, en wat Sherlock *et al.* op 'n meer algemene wyse gebruik. As 'n mens die neuropsigiatriese sindroom na willekeur in ‚voorbewustelose' en ‚bewustelose' toestande verdeel, sal die prognose van die laasgenoemde tipe van pasiënte natuurlik ernstiger wees. 'n Ander punt is die kliniese erkenning van die vroeë ‚voor-bewustelose' toestand; wanneer begin 'n siek pasiënt ‚belemmering van emosionele en geestesintellek' toon?

1. Kirk, E. (1936): Acta. med. scand., suppl. 77.
2. Bessmann, S. P. en Bessman, A. N. (1955): J. Clin. Invest., **34**, 622.
3. Sherlock, S., Summerskill, W. H. J., White, L. P. en Phear, E. A. (1954): Lancet, **2**, 453.
4. Sherlock, S., Summerskill, W. H. J. en Dawson, A. M. (1956): Lancet, **2**, 689.
5. Sherlock, S. (1956): Practitioner, **177**, 446.
6. Gabuzda, G. J. en Davidson, C. S. (1954): Ann. N.Y. Acad. Sci., **57**, 776.
7. Gyorgy, P., Stokes, J., Smith, W. H. en Goldblatt, H. (1950): Amer. J. Med. Sci., **200**, 6.

morrhage; oesophageal compression by means of a swallowed tube may be life-saving.

The Hammersmith results are distinctly better than those obtained in any previous series of cases of liver coma. Of the 39 cases (58%) that recovered, 21 had been in deep coma; in particular, 6 of the 13 cases of viral hepatitis recovered. The previous series reported showed considerably higher mortality figures (the number of cases that recovered, as quoted by Sherlock are shown in parenthesis): Foulk *et al.*, hepatic cirrhosis 52 (5); Katz and Ducci, viral hepatitis 1,000 (*nil*); Stokes *et al.*, viral hepatitis 23 (2); McDermott *et al.*, portal cirrhosis 20 (4). The higher death-rates recorded here may be due to confusion over the definition of 'hepatic coma', which these workers may confine to the description of unconscious patients and which Sherlock *et al.* use in a more general way. If one arbitrarily divides the neuropsychiatric syndrome into 'pre-unconscious' and 'unconscious' states, the prognosis of the latter type of patient will naturally be graver. Another point is the clinical recognition of the early 'pre-unconscious' state; when does a sick patient begin to show 'impairment of emotional and mental intellect'?

1. Kirk, E. (1936): Acta med. scand., suppl., 77.
2. Bessman, S. P. and Bessman, A. N. (1955): J. Clin. Invest., **34**, 622.
3. Sherlock, S., Summerskill, W. H. J., White, L. P. and Phear, E. A. (1954): Lancet, **2**, 453.
4. Sherlock, S., Summerskill, W. H. J. and Dawson, A. M. (1956): Lancet, **2**, 689.
5. Sherlock, S. (1956): Practitioner, **177**, 446.
6. Gabuzda, G. J. and Davidson, C. S. (1954): Ann. N.Y. Acad. Sci., **57**, 776.
7. Gyorgy, P., Stokes, J., Smith, W. H. and Goldblatt, H. (1950): Amer. J. Med. Sci., **220**, 6.

WHAT SHALL WE DO WITH OUR SAMPLES?

After every postal delivery, the harassed practitioner finds himself breasting an ever increasing wave of envelopes, letters, circulars, periodicals, blotting paper and samples. As he manfully brushes aside the one wave, another appears, and the tide flows steadily in. After a while, every doctor develops a special technique for dealing with this part of his mail. Some, more cowardly in nature, hand the whole affair over to their receptionists; others conscientiously plough through the contents of the envelopes, throwing the blotting paper into one drawer and the free samples into another. There they lie, mute evidence of the generosity and business acumen of the pharmaceutical houses. When the drawers are full the overdue house-cleaning takes place, and most of these preparations in their gorgeously covered capsules are consigned to the incinerator or the refuse bin.

It is a sad fact that a really successful pharmaceutical product appears only once on the doctor's free list. The absolutely unsuccessful preparation likewise appears once, and perhaps twice, more in the hope than with any assurance of a friendly reception. The doubtful preparations keep on appearing in our mail. As time goes on the indications for their uses are broadened, the samples become more and more generous, and suddenly one day they disappear, leaving the doctor to cope with numerous bottles, phials or capsules of a preparation that he no longer hears about, has never used, and may be more than a little uncertain of what it is good for. The final step is the disposal of the remnants of these *disjecta membra* of the pharmaceutical houses' fond hopes.

As long as the samples are relatively innocuous, not much harm is done by sending them through the post; but many of the drugs so delivered are potent and some are actually dangerous. How is the doctor to dispose of these samples without danger to those children and adults who may be tempted to taste them or try their effects as medicine?

It would be interesting to know what evidence the pharmaceutical houses have that their products are prescribed because of the unsolicited samples they send us.

CONGENITAL ADRENAL HYPERPLASIA

REPORT OF A CASE IN A MALE INFANT

B. ZILBERG, M.B., CH.B., M.R.C.P.E., D.C.H.

Cape Town

Congenital adrenal hyperplasia is a rare cause of vomiting in infancy. In 1940 Wilkins, Fleischman and Howard[1] published the first report of this condition, in a male child. Despite the paucity of reported cases, it is commoner than is generally realized. Probably many cases are incorrectly diagnosed on account of the confusing clinical picture.

The adrenal cortex produces 3 types of hormone, and hyperadrenocorticism may result in over-production of any one of them. As a result, 3 distinct clinical syndromes may be attributed to adrenocortical overactivity:

1. *The adrenogenital syndrome* is due primarily to hypersecretion of the androgenic hormones and may be either congenital or acquired. The congenital variety is almost invariably the result of adrenal hyperplasia. When it occurs in the female it leads to varying degrees of pseudo-hermaphrodism (female intersexuality) with progressive virilization. In the male it leads to macrogenitosomia praecox. The condition is characterized by an excessive secretion of 17-ketosteroids and, in a considerable percentage of cases, this is accompanied by a deficient secretion of glucocorticoids and mineralocorticoids. When the hyperplasia arises postnatally, virilism results in the female and precocious sexual development in the male.

2. *Cushing's syndrome* is due to an excess of the glucocorticoids and is characterized by obesity of the 'buffalo' type, plethora and purple cutaneous striae, hypertension, hyperglycaemia, and occasionally osteoporosis.

3. *Primary aldosteronism or Conn's syndrome* has recently been recognized and is attributed to an excess of adrenal mineralocorticoids. It is characterized by intermittent muscular pains, cramps, weakness, paralyses and hypertension. Renal dysfunction occurs and tetany may be a feature. The blood shows hypokalaemia, hypernatraemia and alkalosis.

THE CLINICAL PICTURE IN CONGENITAL ADRENAL HYPERPLASIA

The clinical picture is due to an excessive secretion of androgens, modified in many cases by associated glucocorticoid and mineralocorticoid deficiency.

Effects of Excessive Androgen Secretion

In the female infant genital abnormality is recognizable from birth. There is usually enlargement of the clitoris. In severe forms the masculinization is more marked, with labio-scrotal folds and the persistence of a urogenital sinus which receives both urethra and vagina. Thus, in an infant with equivocal sex, congenital adrenal hyperplasia must be considered. Precocious development takes place in a male direction.

In the male excessive genital development may be present at birth, but is usually not noticeable till after 1 year of age. Sexual development progresses rapidly and the penis and prostate may attain adult size at an early age. Erections are frequent, sexual hair and acne appear prematurely, and the voice becomes deep. In spite of the marked development of secondary sexual characteristics, it is noteworthy that the testes usually remain small and immature and spermatogenesis does not occur. The patients, unless they succumb during crises, grow rapidly and become exceedingly muscular. Bone growth is accelerated and epiphyseal fusion occurs early. Thus growth stops prematurely and the child may present the picture of an 'infant Hercules'.

Effects of Glucocorticoid and Mineralocorticoid Deficiency

In many cases (Russell[2] reports 68% in a series of 38 males with congenital adrenal hyperplasia) evidence of mineralocorticoid and glucocorticoid deficiency of the type seen in Addison's disease becomes manifest within the first few weeks of life. The predominant symptom is vomiting, which may be projectile and may closely simulate that seen in pyloric stenosis. Dehydration soon follows, but death may occur before this becomes marked. Other features are diarrhoea, rapid breathing, and episodes of collapse with pallor or cyanosis. Frequently dehydration is out of keeping with the extent of vomiting or diarrhoea. A craving for salt may be a notable feature. The severity of these symptoms is variable. The severest forms simulate 'Addisonian crises' and, unless correctly treated, these crises may soon result in death.

The early recognition of this disease is important, for these patients can lead fairly normal lives if they receive adequate treatment.

Biochemical changes may not be present until the third week or may be found only during periods of crisis. The levels of serum sodium, chloride and bicarbonate fall, while that of serum potassium rises—a chemical picture like that seen in Addison's disease. The diagnosis may be confirmed by demonstration of the excessive urinary excretion of 17-ketosteroids and pregnanediol.

The family history may help in the diagnosis in that siblings are commonly affected or may have exhibited intersexuality.

THE UNDERLYING METABOLIC DEFECT AND ITS BIOLOGICAL EFFECT

In the normal individual the adrenal cortex is under the control of the anterior pituitary *via* its ACTH secretion. ACTH stimulates the adrenal cortex to secrete hydrocortisone and androgens. (The role of the pituitary gland in the control of aldosterone production has not been clearly elucidated.) Circulating hydrocortisone has an

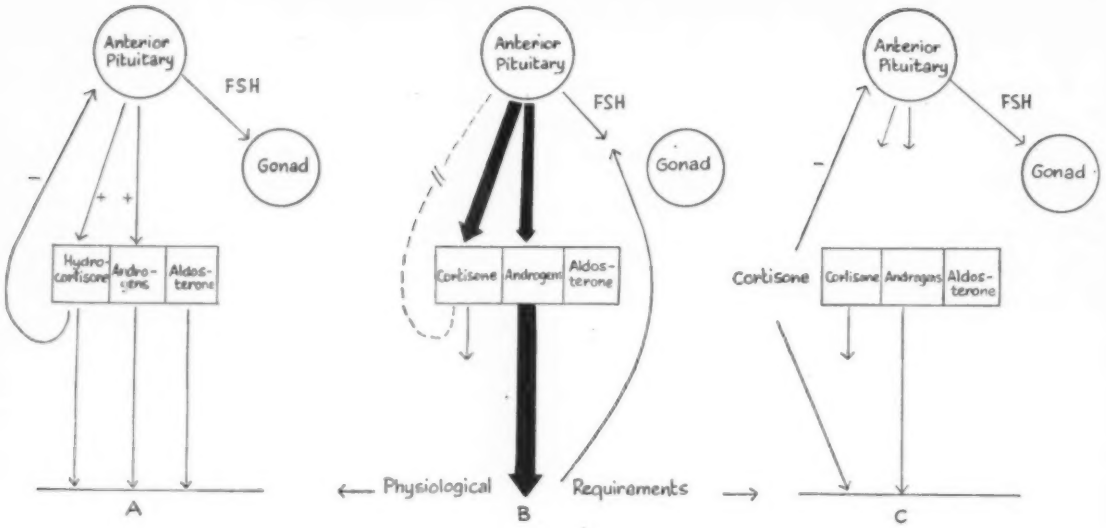

Fig. 1

(*a*) *Pituitary adrenal axis in normal individual.* The anterior pituitary *via* ACTH stimulates the adrenal cortex to secrete hydrocortisone and androgens. The circulating hydrocortisone has an inhibitory effect on the pituitary so that a homeostatic balance is established. *Via* FSH secretion gonadal maturation is stimulated.

(*b*) *Pituitary adrenal axis in congenital adrenal hyperplasia.* Owing to the deficiency of hydrocortisone synthesis, ACTH production is excessive, resulting in over-stimulation of the adrenal cortex, which pours out androgens. FSH is inhibited by the excess of androgens.

(*c*) *The effect of the administration of cortisone in congenital adrenal hyperplasia.* The cortisol deficiency is corrected and inhibition of ACTH production is effected. The adrenal is put to rest and the excessive androgen production is stopped. The 'brake' on FSH is removed, allowing gonadal maturation.

inhibitory effect on the anterior pituitary so that a fine homeostatic balance is established. *Via* FSH production the pituitary is responsible also for gonadal maturation (Fig. 1*a*).

The underlying defect in congenital adrenal hyperplasia is illustrated in Fig. 2. In the course of production of 17-hydroxycorticosterone (compound F, hydrocortisone or cortisol) by the normal adrenal, the last step involves the conversion of 17-hydroxyprogesterone to compound F.[3] Jailer[4] suggests that in congenital adrenal hyperplasia there is an enzyme block at the 17-hydroxyprogesterone stage with consequent excessive accumulation of this compound, which is reflected in the high levels of urinary steroids.

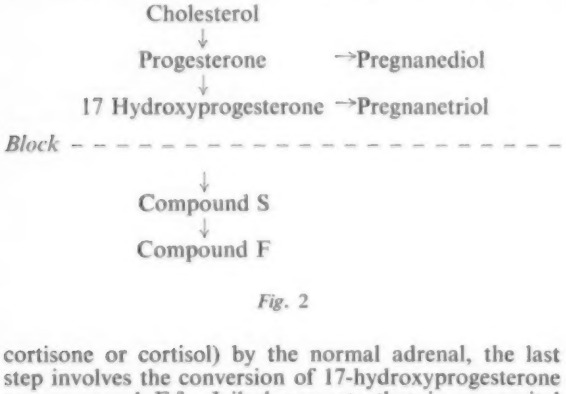

Fig. 2

Thus hydrocortisone precursors accumulate, but there is an absolute deficiency of hydrocortisone itself. This results in uninhibited secretion of ACTH by the anterior pituitary. In its turn this causes the adrenals to hypertrophy and to secrete large quantities of androgens (Fig. 1*b*). In addition, ACTH excess leads to further over-production of abnormal steroids by the adrenals. These abnormal steroids are believed to be androgenic, though conclusive proof of this androgenic activity is lacking.[5]

As a result of over-production of these androgenic substances during intra-uterine life, external genital abnormalities occur in the developing female foetus. If the anomaly arises before sexual differentiation is complete, masculinization of the genital tubercle and urogenital sinus results. The extent of this masculinization varies from case to case. The commonest anomaly of the external genitalia is hypertrophy of the clitoris with a urogenital sinus opening into the perineum (the vagina usually opening into the urethra) and hypertrophied labio-scrotal folds. The severest forms show almost complete external maculinization, but a penile urethra is very rare. The mildest cases demonstrate only hypertrophy of the clitoris.

In the male foetus the changes are less striking; prostatic and penile enlargement may ensue.

The continued excessive production of androgens throughout childhood is responsible for precocious sexual development in the male, and for increasing

virilism with early masculine puberty in the female. But this is not true precocity, since spermatogenesis or ovulation does not occur. This may result from inhibition of FSH production by the large amounts of circulating androgens (Fig. 2*b*).

The deficiency of hydrocortisone which occurs in congenital adrenal hyperplasia is responsible for the metabolic features. Renal tubular function is impaired in the absence of cortisol, with consequent disturbances of water, acid and base metabolism; a tendency to dehydration, acidosis and hyperkalaemia results.

The increased susceptibility and poor response to infection occurring in the syndrome are related to the deficient production of hydrocortisone.

Aldosterone deficiency has not finally been shown to occur in adrenal hyperplasia, but may in part be responsible for the low serum-sodium levels.

When cortisone is administered to patients suffering from the adrenogenital syndrome it has a twofold action: (1) It inhibits ACTH production by its direct action on the pituitary; (2) it provides cortisone for body cells, thereby decreasing the tissue demand for cortisone and thus indirectly inhibiting the production of ACTH. The production of large amounts of androgens is stopped and virilization ceases. The clitoris or penis regresses and, in the female, breast development may now occur at the normal time. The depressant effect of the excessive androgens on FSH production is removed, so that eventual sexual maturation may occur (Fig. 1*c*).

CASE REPORT

In May 1954 a 3-weeks-old infant was admitted to the Birmingham Children's Hospital with a story of projectile vomiting which started a few days after birth. There was no constipation. Pregnancy and labour had been normal. Family history was non-contributory

Physical examination revealed a well formed, rather hirsute, infant with mild peripheral cyanosis. Visible gastric peristalsis was present, but no pyloric tumour was palpable on repeated examinations. The external genitalia were within normal limits and no other abnormal findings were present. Vomiting persisted after admission to hospital.

Special Investigations

Urinary examination and routine examination of the blood revealed no abnormality. No pathogens were isolated from the stools.

Chemical analysis of the blood gave the following results: Serum Na 128 mEq./l., Cl 100 mEq./l., K 8·5 mEq./l., Ca 12 mg.%. CO_2-combining power 40·7 vols.%. Blood urea 50 mg.%. Fasting blood-sugar 100 mg.%.

Twelve hours later the chemical results were as follows: Serum Na 130·5 mEq./l., Cl 110·6 mEq./l., K 10 mEq./l., CO_2-combining power 16·5 vols.%.

An electrocardiogram showed tall, peaked T waves in most leads, supporting the biochemical diagnosis of hyperkalaemia.

Course and Treatment

At this stage the patient's clinical condition deteriorated. He was drowsy and dehydrated, the limbs were hypotonic, and tendon reflexes could not be elicited. The pulse rate and rhythm were normal.

Intravenous fluids were started and intake and output balances were kept. M/6 solution of sodium lactate was used to overcome the acidosis. Cortisone unfortunately was not available at this stage; 2 mg. of desoxycorticosterone acetate (DCA) was given intramuscularly. Correction of acidosis and hyponatraemia tends to lower serum-potassium levels. But these levels were considered to be dangerously high in this infant and more specific potassium-lowering measures were instituted, as follows: (1) Rapid lowering of the potassium level was attempted by the intravenous administration of 10% dextrose solution (20 ml. per lb. body-weight). At the same time 5 units of insulin was given subcutaneously and 4 c.c. of a 10% solution of calcium gluconate intravenously. (2) In an effort to produce a more permanent lowering of the potassium level, 1,600 mg. of 'Rezonium A', a cation-exchange resin with selective action on the K^+ ion, was given by stomach tube at 8-hourly intervals.

The intravenous administration of 'Rezonium A' and DCA was continued for 4 days, and the patient's clinical condition was much improved; 'blood chemistry' was now within normal limits. The patient was placed on oral dried-milk feeds with 2 g. of salt added daily. DCA, 2 mg. daily, was continued.

Four days after cessation of intravenous fluids, vomiting and dehydration recurred. The results of chemical examination were then: Serum Na 130·5 mEq./l., Cl 107·1 mEq./l., K 11·6 mEq./l., CO_2-combining power 33·3 vols.%. Balance studies showed negative Na^+ and Cl^- balance despite DCA and additional salt.

The results of estimations of urinary 17-ketosteroid were now available; they showed values of 5 mg. and 3 mg. per 24 hours on 2 successive days. These high values confirmed the diagnosis of adrenal hyperplasia.

Intravenous therapy was started again and the original therapeutic regime was followed. In addition, 25 mg. of cortisone daily was given by intramuscular injection. When thirsty, the patient was offered normal saline, which he drank with relish. After 48 hours the clinical condition improved and the results of chemical examination of the blood were normal. Intravenous therapy was discontinued and dried-milk feeds with 3 g. of salt per day were given. In view of a constant tendency to acidosis, 1 g. of NaCl was later replaced by an equivalent amount of M/6 solution of sodium lactate. An attempt to withdraw the DCA resulted in dehydration; this necessitated correction with subcutaneous infusions and DCA substitution was therefore continued.

Gradually the additional salt and sodium lactate were removed from the feed. Cortisone, 6·5 mg. 6-hourly, was administered orally. DCA was replaced by the long-acting depot preparation Percorten 17 (Ciba) (50 mg. of the crystalline preparation) being given every 4 weeks. 17-Ketosteroid excretion was now maintained at less than 1 mg. per 24 hours.

The patient was discharged on a regime of oral cortisone and depot DCA. His mother was advised to add salt to his feed should vomiting recur. In view of the liability to crises, return to hospital was advised in the event of any infective illness.

The case was followed up for 18 months, at which time the patient appeared to be developing normally; the bone age and 17-ketosteroid excretion were within normal limits.

DISCUSSION OF DIAGNOSIS

In this child the early occurrence of vomiting and crises of collapse with dehydration associated with acidosis, hyperkalaemia and hyponatraemia, strongly suggested the diagnosis of congenital adrenal hyperplasia. This diagnosis was confirmed by the finding of 24-hour 17-ketosteroid excretions of 5 mg. and 3 mg. on two successive days, the normal value for this age being less than 0·6 mg. per day.[2]

Differential Diagnosis

Other conditions which had to be considered in this case were:

1. *Congenital hypertrophic pyloric stenosis.* This was excluded by the very early onset of vomiting, the normal bowel actions, the absence of a palpable tumour, and the normal barium meal. The biochemical disturbance in pyloric stenosis is usually that of alkalosis. Other forms of intestinal obstruction were excluded by the absence of constipation and by the barium studies.

2. *Hiatus hernia* was excluded by the type of vomiting, the absence of haematemesis, and the barium swallow.

3. *Renal abnormality*. There were no urinary symptoms; and the absence of a renal mass, the normal composition of the urine, and the relatively low blood-urea (50 mg.% in the presence of dehydration), argued against a renal abnormality as the cause of the acidosis and hyperkalaemia.

DISCUSSION OF TREATMENT

Treatment aims at suppressing excessive production of androgen, replacing the hormonal deficiencies, and maintaining normal electrolyte balance.

In 1950 it was shown by Wilkins *et al.*[6] that cortisone in congenital adrenal hyperplasia suppresses the excessive secretion of urinary 17-ketosteroids and biologically active androgens. It prevents the virilizing effects of the androgens and the accelerated growth and premature fusion of the epiphyses. It also has a slight sodium-retaining effect and thus also helps in correcting the disturbed electrolyte state. The dual action of cortisone, (1) as a replacement measure and (2) as a 'brake' on excessive production of ACTH by the anterior pituitary, is diagrammatically shown in Fig. 1*c*. Initial suppression of androgen is effected by intramuscular cortisone in a dosage, for infants, of 25 mg. daily. After 5-10 days this dosage may need readjustment. Oral dosage is usually 2-3 times as high.[7] Adequacy of dosage is gauged by maintenance of 17-ketosteroid excretions at levels roughly appropriate to the age of the child, and by radiological evidence that bone growth is proceeding at the normal rate. The requirement of cortisone may diminish after a while and it may be possible to decrease the frequency of administration. In some patients it may be advisable to give weekly injections of slightly larger doses of cortisone.[8]

Cortisone alone will not control the loss of sodium in all patients. When it does not, additional salt up to 7 g. daily is given. If balance is still not maintained DCA is added.

During infections the amounts of salt and DCA must be increased, and in some cases increased dosage of cortisone may also become necessary.

Compound B (corticosterone) has marked sodium-retaining properties, and this may well be the ideal corticoid to use in the patient who shows a marked tendency to lose salt.

Management during Crisis

Replenishment of the sodium deficit is accomplished by the intravenous route.

Before intravenous cortisone became available reliance was placed on aqueous extracts of adrenal gland, up to 20 ml. a day being given in the infusion. Intramuscular cortisone was started at the same time. The use of the recently available intravenous preparation of hydrocortisone may prove life saving.

In our patient the diagnosis was first made during a period of crisis. The serum-sodium levels, however, did not drop low enough to warrant the intravenous use of aqueous glandular extracts, with the attendant danger of pulmonary oedema.

Other Measures

The very high potassium levels may require more rapid lowering than is effected by cortisone. Immediate treatment of hyperkalaemia consists of the intravenous injection of 5% calcium gluconate (between 0·5 and 3·0 ml per lb. body-weight and of 10% dextrose (20 ml. per lb.), and the subcutaneous injection of 5 units of insulin. Insulin and dextrose, by stimulating glycogenesis, accelerate the migration of potassium from the plasma into the cells. Calcium antagonizes the toxic effect of potassium on the myocardium. The cation-exchange resins may prove a useful adjunct to potassium-lowering measures. These act by extracting 'unwanted' ions and replacing them by ions contained within the structure of the exchange substance. 'Rezonium A' has sodium occupying the exchange site and the K^+ ion is withdrawn from the blood and replaced by Na^+ when Resonium is used in states of hyperkalaemia. It is suspended in a mucilage and given by gastric tube. The suggested dose for an infant is 600 mg. per lb. per day, given in 3 divided doses. It may also be used as a retention enema. Regular biochemical control is essential when these measures are employed.

SUMMARY

A case is described of congenital adrenal hyperplasia in a 3-weeks-old male infant presenting with 'Addisonian' crises.

The characteristic combination was present of vomiting, hyperkalaemia, hyponatraemia, dehydration, acidosis and excessive androgen-production.

The severe metabolic disturbance was corrected by the use of cortisone and long-acting DCA.

An interesting feature was the craving for salt shown by the patient during periods of crisis.

The pathogenesis of the condition and the mode of action of cortisone in controlling its features are discussed.

I am indebted to Dr. C. Smallwood of the Birmingham Children's Hospital for permission to publish this article, and to Dr. R. Hoffenberg, of Groote Schuur Hospital, Cape Town, for his help in its preparation.

REFERENCES

1. Wilkins, L., Fleischman, W. and Howard, J. E. (1940): Endocrinology, **26**, 385.
2. Russell, A. (1954): *Recent Advances in Paediatrics*, p. 262. London: Churchill.
3. Bongiovanni, A. M. (1953): Bull. Johns Hopk. Hosp., **92**, 244.
4. Jailer, J. W. (1955): *Adrenal Function in Infants and Children*. Report of the XIII M & R Pediatric Research Conference, p. 53. Columbus, Ohio: Ross Laboratories.
5. Reifenstein, E. C. (1956): J. Clin. Endocr., **16**, 1262.
6. Wilkins, L., Lewis, R. A., Klein, R. and Rosenberg, E. (1950): Bull. Johns Hopk. Hosp., **86**, 249.
7. Wilkins, L. (1952): J. Pediat., **41**, 860.
8. Segaloff, A., Gordon, D. and Horwitt, B. N. (1955): J. Amer. Med. Assoc., **157**, 1479.

SECONDARY ABDOMINAL PREGNANCY

REPORT OF A CASE IN A BANTU FEMALE

J. L. SMITH, M.B., CH.B. (CAPE TOWN)

and

E. C. NEWBORN, M.B., B.CH. (RAND)

Matatiele, East Griqualand

It is well established that the fertilised ovum may become implanted not only in the uterus, but in the fallopian tube or in the ovary; whether its implantation upon the peritoneum as a primary event can occur has been discussed but never proved. It is known, however, that secondary abdominal pregnancy occurs, and then it always is secondary to a tubal pregnancy.

When the ovum lodges in the fallopian tube it is always the result of an accident. The predisposing factors comprise many mechanical conditions relating to the tubes and some authorities include anomalies of the fertilized ovum in the etiology of tubal pregnancy. A tubal gestation may terminate in many different ways but we are now concerned only with the event where the ovum escapes from its cramped surroundings and pursues its development under more favourable conditions. This usually follows an external tubal haemorrhage and spontaneous tubal rupture.

Rupture of the fallopian tube may be intraperitoneal, with haemorrhage into the abdominal cavity resulting in death from haemorrhage or in (*a*) retro-uterine or pelvic haematocoele with death of the foetus, (*b*) secondary tubo-abdominal pregnancy, or (*c*) secondary abdominal pregnancy.

Rupture of the tube may also be extra-peritoneal and here there may be death of the foetus and broad-ligament haematoma, or the foetus may survive and an intra-ligamentary pregnancy may occur.

For the purposes of this discussion we shall consider secondary abdominal pregnancy only where intra-peritoneal rupture or tubal abortion has occured. This usually results when the muscle and peritoneum of the fallopian tube has been eroded and the external boundary of the ovum is composed only of trophoblast and fibrinous tissue. The distension of the tube caused by the growing foetus and the eroding action of the villi have been the predisposing factors. The commonest time for this rupture to occur is between the 8th and 10th weeks, but it may be earlier or later than this. If the rent which occurs is on the roof or on the sides of the tube, the rupture will involve the peritoneal covering, and the blood effused will be poured out into the general peritoneal cavity. The ovum may or may not be completely expelled with it. Usually when this happens death of the foetus occurs and we are left to deal with a 'ruptured ectopic' as a surgical emergency.

Occasionally, however, the ovum may continue its development. The placenta becomes attached to neighbouring peritoneal surfaces, while layers of lymph are deposited upon the exposed amnion from the surrounding peritoneum, forming a false membrane which constitutes a secondary gestation sac. This secondary sac becomes further strengthened by adhesion to the neighbouring peritoneal surfaces, including omentum, coils of intestine and the abdominal parieties. And so the gestation sac may be strengthened and the placental blood supply augmented by adherent omentum and mesentery.

The pregnancy may continue to term. The patient then usually passes through a 'false labour,' which is interesting in that, although attended by severe cramp-like pain, it is not associated with uterine contractions. This false labour always leads to the death of the foetus, but why this should be is unexplained.

If the foetus is retained within the abdominal cavity and the gestation sac remains uncomplicated by infection or haemorrhage, eventually the foetus shrinks by absorption of its fluid constituents. Upon the dried tissues lime salts become freely deposited, convering it into a litho-paedion.

Clinical Features. The recognition of this condition presents considerable difficulties. The history of the pregnancy usually has some abnormal features such as attacks of lower abdominal pain in the early months, accompanied by some haemorrhage. When the ovum survives a tubal rupture the amount of internal bleeding appears to be inconsiderable and the accompanying symptoms of a ruptured ectopic are usually less urgent.

On examination, if the pregnancy is well advanced it will be found that the cervix is barely soft and the uterus is small and displaced by a mass which is distinct from it.

CASE HISTORY

C.M., a Native female aged 34 years, was admitted to hospital on 15 September 1956. She complained of severe lower abdominal pain associated with vaginal bleeding, which had been present for the past month. The last menstrual period had been in June. At no time previous to the onset of her symptoms had there been an acute abdominal episode and she had never fainted or vomited. As far as she was aware no products of conception had been passed vaginally.

She had had 5 pregnancies, 3 of which had proceded to full term and 2 had been abortions in the early months of pregnancy. Only one child was alive, aged 15 years. The last pregnancy had been a full-term one, the child having been born in November 1955 and died at the age of 3 months from gastro-enteritis.

The patient was found to be an extremely ill, thin woman with a temperature of 102°F. She was very anaemic, with a dark-brown haemorrhagic vaginal discharge. Vaginal examination at this stage was difficult, owing to board-like rigidity of the lower abdomen. The findings, however, were a hard cervix with no fullness in either fornix or in the pouch of Douglas; the cervix was not tender on movement; the fundus of the uterus could not be felt.

Although a ruptured ectopic pregnancy was considered, it was thought to be unlikely in view of the history and physical signs elicited. The most likely diagnosis appeared to be a lower abdominal inflammatory lesion.

Owing to the patient's general condition surgery was considered unwise. Restorative and antibiotic therapy was instituted. She was given a transfusion of 2 pints of blood, but she remained anaemic and ill. Ten days after admission it was possible to establish the following facts:

A small non-pregnant retroverted uterus was palpable. Separated from the fundus of the uterus by a sulcus of approximately 1 inch in width was a fairly hard mobile mass, measuring 6 by 3 inches, with its long axis lying transversely. The relationship of this mass to any other organ could not be established. The diagnosis remained obscure. The patient's condition remained too poor to permit of operative interference until, after a further period of restoration, she underwent a laparotomy on 6 October.

At operation about 200 c.c. of free blood was found in the peritoneal cavity. Slightly to the right of the mid-line, attached to loops of jejunum and covered by adhesions, was a tubular mass, greyish-blue in colour. On closer inspection a gestation sac and placenta was seen. In endeavouring to separate the sac from its surrounding structures the covering membrane was ruptured and a foetus of about 16 weeks gestation, attached by its umbilical cord to a posteriorly situated placenta, was found.

In attempting to separate the mass from its attachments haemorrhage was caused by inadvertently tearing portions of placenta. It was therefore considered that the only way in which to proceed was to remove the gestation sac and foetus by ligation of its cord. The placenta was left *in situ* and haemorrhage was controlled by packing and ligation of obvious bleeding vessels. Inspection of the uterus, tubes and ovaries revealed no abnormalities. Eventually the bleeding was completely controlled and the abdomen closed without drainage.

Throughout the operation the patient received blood transfusion. Her immediate post-operative condition was extremely poor and it was feared that she would not survive. However, possibly owing to the fact that she had become fairly well compensated to an anaemic state, she not only survived but improved rapidly. She had no post-operative upsets and was discharged from hospital on the 10th post-operative day, to all intents and purposes perfectly fit, although still anaemic.

DISCUSSION

This case presented the clinical features of a secondary abdominal pregnancy, but they were not realized. Owing to the fact that the patient continued to bleed *per vaginam* and that a mass was present in the hypogastrium operative interference was considered a necessity. It is possible the pregnancy may have continued if left alone. However, in view of the continued vaginal bleeding, it may be argued in retrospect that the foetus was dead. As the pregnancy had obviously become complicated by infection, the method of dealing with this particular case appears to have been justified.

Complete removal of the placenta in these cases is dangerous, owing to the close attachment to surrounding structures, and the likelihood of causing uncontrollable haemorrhage or a fistula is very real. The placenta is best left alone. Apparently the placenta is always absorbed, at times taking as long as 2½ years in the process. A complication which may be expected is adhesion formation leading to intestinal obstruction.

SUMMARY

A case of secondary abdominal pregnancy is described and the pathology and clinical features of the intra-abdominal type is discussed. The treatment adopted in this case is also briefly outlined and discussed.

REFERENCE

MacLeod, D. H., and Read, C. D. (1955): *Gynaecology*, 5th ed., pp. 178-206. London: Churchill.

CHRONIC THYROTOXIC MYOPATHY

A REPORT OF TWO CASES

R. HOFFENBERG, M.B., CH.B., M.R.C.P.

and

L. EALES, M.D., M.R.C.P.

From the Department of Medicine, Groote Schuur Hospital, Cape Town, and University of Cape Town

Loss of weight is one of the cardinal symptoms of thyrotoxicosis. General muscular wasting forms an integral part of this process and is, at times, very striking. Very different in character is the local disorder of muscle with severe wasting and profound weakness, which occasionally attends thyroid disease.

Starling[1] in 1938, suggested the following classification of these localized muscular disorders:

1. Exophthalmic ophthalmoplegia
2. Thyrotoxic myopathy: (*a*) acute, (*b*) chronic
3. Thyrotoxic periodic paralysis
4. Myasthenia gravis associated with thyrotoxicosis.

Acute thyrotoxic myopathy is said to be characterized by rapidly progressive weakness involving the bulbar, limb and trunk muscles, with a fatal termination in one or two weeks following respiratory paralysis. This syndrome is not universally accepted as a separate entity,[2] since the few cases reported cannot readily be distinguished from myasthenia gravis occurring in association with thyrotoxicosis.

Chronic thyrotoxic myopathy is characterized by severe muscular atrophy, which frequently involves only a few muscle groups and which often overshadows the other manifestations of thyrotoxicosis. Such disproportionate involvement of an organ or a system in thyrotoxicosis is well recognized. It is perhaps best demonstrated in the so-called 'masked thyrotoxic' cardiac case, where the cardiac features dominate the clinical picture.

In this paper we report two cases of chronic thyrotoxic myopathy in which complete recovery took place.

CASE REPORTS

Case 1

J. de K., White male 49 years old (Fig. 1). After a prolapse of the rectum in April 1953, this patient had experienced loss of strength and energy, tremor, increased sweating, irritability and nervousness, dyspnoea and palpitation on exertion. His weight had dropped from 142 lb. to 128 lb. A prominent symptom at this time was pain, stiffness and weakness, affecting the thigh muscles.

Fig. 1. Case 1. Note extreme emaciation.

In July 1953 a diagnosis of thyrotoxicosis was made (a single BMR reading was said to have been +85%). The administration of methyl thiouracil resulted in gain in weight to 141 lb., symptomatic improvement and slowing of the pulse. Treatment was discontinued in October 1953 because of marked enlargement of the thyroid gland.

In April-May 1954 for one month, the patient had painful, tender, red swellings involving the wrists, metacarpo-phalangeal and proximal interphalangeal joints of both hands and the right knee-joint. Cortisone (dose unknown) was given for 2 weeks, with improvement in the joints. By June 1954 his weight had dropped to 117 lb. and there was, once again, pain and weakness of both thighs, with the development of contractures involving the knees and some of the hand joints. Oral hydrocortisone was given for 2 weeks with no improvement. Thereafter, his weight continued to fall despite a good appetite; the patient lost strength and became shaky; dyspnoea and palpitation recurred.

In January 1955, he was admitted to Groote Schuur Hospital for the first time.

On examination he was found to be extremely wasted (97 lb.). The eyes were staring, but showed no lid-lag, lid-retraction or exophthalmos. A firm, symmetrical thyroid enlargement was present. There was gross wasting of the small muscles of the hand with fixation of several joints and Dupuytren's contractures. Ulnar deviation was present. The knees were partially flexed owing to contracture of the posterior thigh-muscles and there was very gross wasting of these and the anterior group. The wasted muscles were not tender and the skin was normal. Motor power was markedly diminished. The tendon reflexes were normal.

The pulse varied from 90 to 120 beats per minute (the sleeping pulse-rate exceeded 90). There was no tremor; palmar erythema was present, but the palms were not unduly warm or damp.

Haemoglobin 16·5g.%. Leucocyte count 9,700 per c.mm.

E.S.R. 28 mm. in 1st hour (Westergren).

Routine urine examination showed no abnormality.

Special Investigations

X-rays of the hands and knees showed demineralization around the affected joints. The chest film revealed scattered calcified shadows compatible with healed pulmonary tuberculosis.

The electrocardiogram was normal apart from tachycardia.

Serum sodium 138·7, potassium 4·4, chloride 103 mEq./l.

Serum CO_2 combining power 66 vols.%.

Serum calcium 10·1, inorganic phosphorus 4·3 mg.%.

Serum alkaline phosphatase 9·5 units (Bodansky).

Serum albumin 3·8, globulin 1·7 g.%.

Blood urea 29 mg.% on 28 January, 29 mg.% on 16 February 1955.

Serum uric acid 4·5 mg.%.

Thymol turbidity 1, thymol flocculation 0.

Serum cholesterol 177 mg.% (1 February), 194 mg.% (4 March), 198 mg.% (1 April), 204 mg.% (14 May 1955).

Glucose tolerance test: Fasting blood-sugar 73 mg.%; (50 g. of glucose given by mouth) after one hour 150 mg.%, after two hours 125 mg.%.

Urea clearance test (maximum): 1st period 152·5% standard, 2nd period 151·5% standard.

Urine calcium 261 mg./day on 6 February, 212 mg./day on 7 February 1955.

24-hour urine creatinine/creatine excretion (average of several days' collection) (see chart in Fig. 2): 9-11 February 830/350 mg., 24-28 February 1,080/255 mg., 3 March 1,180/200 mg., 18 March 1,010/125 mg., 2 April 800 mg./nil 30 April-3 May 1,020 mg./nil.

Blood Wassermann and Berger reactions negative.

B.M.R.: +32%, +22%, +12% (on successive days before institution of therapy).

Thorn test: Normal eosinophil response to 8-hour intravenous infusion of ACTH.

Neostigmine test: No improvement following 0·5 mg. of neostigmine intravenously.

Lumbar puncture: Normal hydrodynamics and CSF.

Muscle Biopsy: There was some variation in thickness of muscle fibres, with focal areas of sarcolemmal nuclear proliferation; one such area was seen around a necrotic muscle fibre.

No facilities were at hand for radioactive iodine tests or estimation of protein-bound iodine.

Course

On 24 February propyl thio-uracil was commenced in a dose of 200 mg. t.d.s. This was reduced to 100 mg. t.d.s. after 2 weeks.

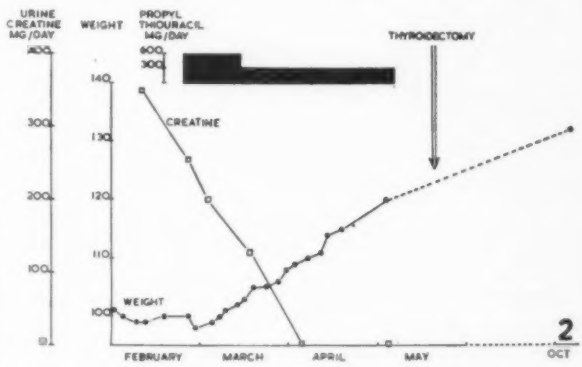

Fig. 2. Case 1. Chart showing response to therapy.

The response which ensued, while not dramatic, was steady and maintained. Within 8 weeks weight had risen from 97 to 114 lb., the tachycardia had resolved, and urine creatine had dropped progressively from the initial reading of 350 mg./day to *nil* on 2 April (see chart in Fig. 2). *Pari passu* with these observed improvements, strength and muscle bulk returned and general symptomatic well-being was restored.

During this therapy the thyroid gland enlarged (maximal neck circumference rose from 13½ inches to 15¼ inches). Laevothyroxine, 0·1 mg. t.d.s., was administered and reduction in the size of the gland was noted.

On 20 May 1955 subtotal thyroidectomy was performed (Prof. J. H. Louw). The operation passed off uneventfully. Histology of the excised gland showed 'acini lined by low cuboidal epithelium containing thin, pale-staining, eosinophilic colloid. The acini also showed intraluminal papillary projections and, in many, evidence of active resorption of colloid'. These changes were considered to be consistent with diffuse toxic hyperplasia which had been treated.

The patient was discharged on 5 June and subsequent examinations have shown continued improvement in muscular strength and general health. The weight rose steadily after operation to 132 lb.

On his most recent visit (October 1955, 6 months after thyroidectomy) he was in excellent health and had resumed his previous occupation. The only complaint was mild stiffness in the fingers.

Case 2

Mrs. I.R., White female 53 years old. For about 8 months the patient had been aware of general weakness, tiredness and loss of energy; there was increased nervousness, increased sweating, decreased tolerance for warm weather, and palpitation. She had lost 50 lb. in weight, despite an increase in appetite, and had noticed prominence of the eyes with a gritty sensation on closing the lids. Apart from the general weakness she felt particularly weak in the legs, and for some months had been unable to raise herself from a sitting position or to climb stairs.

A few months before admission to hospital a 4-week course of methyl thio-uracil had induced amelioration in her symptoms, with return of strength and vigour. This improvement persisted for about a month after cessation of therapy.

She was admitted to Groote Schuur Hospital on 4 April 1956. Mild thyrotoxicosis was present clinically, as evidenced by warm moist palms, a fine tremor of the outstretched fingers, and tachycardia of the order of 85-90 beats per minute.

The eyes showed bilateral exophthalmos with increased resistance to pressure over the eyeballs. There was lid-lag and lid-retraction, limitation of external-rectus movement and diplopia on looking upward and to the right. The conjunctivae were injected and oedematous.

The thyroid gland was firm, nodular, and asymmetrically enlarged; there were no signs to suggest malignancy.

Generalized muscular weakness was present, but the muscles of the pelvic girdle were disproportionately weak. The patient could not raise her legs while lying in bed and was incapable of rising from a supine position on the ground. Her efforts to achieve the latter movement were reminiscent of those seen in pseudo-hypertropic muscular dystrophy. All the leg muscles were wasted, especially the quadriceps. No fasciculation or fibrillation was seen. Deep reflexes were normal and there was no sensory loss.

Over the anterior and lateral aspects of the tibiae there were firm, reddish, raised areas of thickened subcutaneous tissue suggestive of pretibial myxodema. Some lesions formed well-defined, elevated plaques.

Comment. While thyrotoxicosis was unquestionably present, it must be stressed that its clinical manifestations were far from severe. The signs of ocular involvement and, in particular, the muscular wasting and weakness were quite disproportionate to the degree of manifest toxicity.

Investigations

Blood Wassermann and Berger reactions negative.
B.M.R.: +14%; +12%.
Serum cholesterol 136 mg.%.
Serum albumin/globulin: 3·7/2·7 g.%.
Blood urea 25 mg.%.
Thymol turbidity and flocculation: 1 and 0.
Lumbar puncture: Normal dynamics and pressure. CSF normal chemically and microscopically.

Neostigmine test: 2 separate tests, after 0·5 mg. of neostigmine intravenously, produced no alteration in muscular power. 24-hour urine creatinine/creatine excretion: 10 April 460/285 mg., 18-21 April 840/160 mg., 22-24 April 470/245 mg.

Skin biopsy (taken from a thickened plaque over R. tibia). There was deposition of an acid mucopolysaccharide in keeping with that seen in pretibial myxoedema; the corium showed increased vascularity and negligible inflammatory cell infiltration; there was destruction of collagen and elastic fibres, around which the mucin had particularly aggregated.

Course

On 26 April therapy was instituted with methimazole, 20 mg. twice daily; in addition, in view of the hyperophthalmopathia, laevo-thyroxine, 0·1 mg. t.d.s., was administered with the hope of inhibiting endogenous pituitary thyrotrophin production.

Immediate and striking response followed this treatment. The weight rose from 137 lb. to 145 lb. after 10 days of treatment and 152 lb. after a month. The dose of methimazole was gradually reduced as control of the thyrotoxicosis was achieved.

There was regression of all symptoms, the most dramatic evidence of which was the increase of muscle power in the legs. Within 4 weeks of starting therapy the patient was able to walk up 4 flights of stairs without assistance and with no undue fatigue, and she was able to stand up from a supine position rapidly and easily.

All the thyrotoxic signs gradually abated. The eyes improved slowly; the 'gritty' sensation disappeared, proptosis diminished, and the eyeballs felt less tense; conjunctival injection was reduced. Simultaneously the thyroid gland became smaller, until by the first week of June it was no longer visibly enlarged.

Measurements undertaken by the Department of Physiotherapy showed marked increase in the power of the individual muscle-groups. Measurements of thigh and calf circumferences showed remarkable increment.

On 4 June methimazole was stopped and Lugol's iodine was given pre-operatively. Partial thyroidectomy on 22 June was followed by continued improvement. Histology of the excised gland was that of a multi-nodular goitre with no evidence of malignancy.

DISCUSSION

Chronic thyrotoxic myopathy is a rare condition, as judged by the paucity of cases in the medical literature.[2-6] Kite *et al.*,[3] in 1954, reviewed the clinical features of the condition from an analysis of 43 recorded instances of the disease. They excluded many other cases because of inadequate data, but did not include the apparently acceptable patients, 9 in number, reported by Millikan and Haines.[2]

The majority of patients were male, usually in middle life. The onset was gradual, with slow progression of symptoms over months or years. Predominant were loss of weight, muscular weakness and atrophy. Severe muscle cramps were frequently experienced. The muscles of the shoulder and pelvic girdles were maximally affected, with lesser involvement of peripheral muscles; fasciculation was not uncommon, and the majority of patients showed preservation, or even increase, of deep tendon reflexes. Thyroid enlargement was present in most patients, as was a rapid, fine tremor. Eye signs suggestive of hyperthyroidism were found in about 1/3rd of the total number. Other manifestations of thyroid over-activity were not prominent. The majority showed a raised BMR; a minority exhibited a high urinary excretion of creatine. In the few cases reported in recent years in which radio-active iodine tests and serum protein-bound-iodine examinations were performed, these tests confirmed hyperthyroidism.[2] Occasionally a limited response to neostigmine injection was noted. All the patients who had been followed up showed complete recovery after treatment of the hyperthyroidism.

DIFFERENTIAL DIAGNOSIS

A disorder characterized by such severe weakness and wasting may be confused with numerous conditions,

especially since the underlying hyperthyroidism is often difficult to appreciate clinically.

Progressive muscular atrophy develops in a similar age-group, and the clinical picture may be dominated by loss of weight, muscle wasting with fasciculation, and gross weakness. However, the hands and feet are commonly affected in progressive muscular atrophy, rarely in thyrotoxic myopathy. Bulbar palsies are more frequently seen in progressive muscular atrophy and upper-motor-neurone signs are generally present.

Recently attention has been paid to the condition of polymyositis,[7, 8] which appears to be related to dermatomyositis but which may not display the dermal lesions. Adult cases resemble the facio-scapulo-humeral or girdle type of muscular dystrophy.[9] A group has been described in which a rheumatoid type of arthritis occurs. Muscle biopsy may be necessary to distinguish this from thyrotoxic myopathy, although even this may fail to provide the diagnosis in chronic cases. Electromyography is of value. ACTH or cortisone therapy helps many cases of polymyositis, while a therapeutic trial with an antithyroid drug provides a useful diagnostic criterion in thyrotoxic myopathy.

The muscular dystrophies may be separated on the basis of distribution of weakness, family history and lack of response to antithyroid treatment.

Recently a form of myopathy associated with carcinoma has been recognized. This may be a pure myopathy or may be part of a collagen disease such as dermatomyositis.[10–12] The carcinoma may reside in ovary, breast, stomach, kidney, and many other tissues. In some cases removal of the carcinoma is followed by improvement in the neurological picture.

Finally, considerable attention has been paid to myasthenia gravis and thyrotoxicosis occurring in the same patient,[4, 13, 14] but the reported cases of this association are very few in number. In both conditions ocular palsies, creatinuria and lymphocytosis may occur. But they may be distinguished on other grounds: muscle atrophy is prominent in thyrotoxic myopathy, not in myasthenia gravis; weakness is more marked at the end of the day in myasthenia gravis; drooping of the eyelids, as opposed to lid-retraction, occurs in myasthenia gravis; bulbar palsies are common in myasthenia gravis, unusual in chronic thyrotoxic myopathy; and the response to neostigmine, while marked in myasthenia gravis, is absent, or at best limited, in thyrotoxic myopathy. Response to antithyroid treatment is a valuable point diagnostically. In thyrotoxics with associated myasthenia gravis, the myasthenic features may remit on such therapy although, conversely, a 'see-saw' relationship has been described, the myasthenia worsening as the thyrotoxicosis is brought under control, and *vice versa*.[13]

The Diagnosis in our Patients

Despite the lack of confirmation by serum protein-bound-iodine estimation or radio-active-iodine tests, there can be little doubt that our patients suffered from thyrotoxicosis. The inordinate amount of muscle wasting and weakness common to both patients is the basis for the diagnosis of thyrotoxic myopathy. Both patients exhibited other features of interest:

Case 1 presented with gross weight-loss and muscular weakness. The evidence for thyrotoxicosis was not marked. His course had been punctuated by an episode of arthritis which has left residual contractures. The muscular wasting which was present was far too gross to be explained on an arthritic basis and had, in any case, preceded the development of the arthritis by about a year. The extreme weight-loss suggested neoplastic disease, of which the arthritis and myopathy might have been the presenting features. However, the striking response to therapy renders this diagnosis unlikely.

Case 2 showed the remarkable association of thyrotoxicosis, chronic thyrotoxic myopathy, hyperophthalmopathia, and pretibial myxoedema. Her response to antithyroid therapy was equally gratifying.

SUMMARY

Two cases of chronic thyrotoxic myopathy are presented. The diagnostic features of the condition are reviewed and the differential diagnosis is briefly discussed.

We are pleased to acknowledge our thanks to Prof. F. Forman and Dr. S. Berman for their advice and interest; to Prof. J. F. Brock for permission to present case 1, who was under his care; to Prof. J. H. Louw and Mr. R. Lane Forsyth, under whose care case 2 was admitted; to Prof. G. Linder for the biochemical estimations; to Dr. C. J. Uys and Dr. G. Selzer for the pathological reports; to Mr. B. Todt for the photographs; and to Miss M. Lloyd for preparation of the chart.

REFERENCES

1. Starling, H. J., Darke, C. S., Hunt, B. W. and Brain, W. R. (1938): Guy's Hosp. Rep., **88,** 117.
2. Millikan, C. H. and Haines, S. F. (1953): Arch. Intern. Med., **92,** 5.
3. Kite, W. C., Jr., McClintock, J. C. and Graves, R. W. (1954): N.Y. St. J. Med., **54,** 1613.
4. Prasad, R. and Levi, J. E. (1955): J. Clin. Endocr., **15,** 476.
5. McEachern, D. and Ross, W. D. (1942): Brain, **65,** 181.
6. Thorn, G. W. and Eder, H. A. (1946): Amer. J. Med., **1,** 583.
7. Adams, R. D., Denny-Brown, D. and Pearson, C. M. (1953): *Diseases of Muscle: A Study in Pathology.* New York: Hoeber.
8. Walton, J. N. and Nattrass, F. J. (1954): Brain, **77,** 169.
9. Nattrass, F. J. (1956): Proc. Roy. Soc. Med., **49,** 105.
10. Cottell, C. E. (1952): Amer. J. Med. Sci., **224,** 160.
11. Curtis, A. C., Blaylock, H. C. and Harrell, E. R. Jr. (1952): J. Amer. Med. Assoc., **150,** 844.
12. Lansbury, J. (1953): Ann. Rheum. Dis., **12,** 301.
13. Maclean, B. and Wilson, J. A. C. (1954): Lancet, **1,** 950.
14. Cohen, B. (1946): S. Afr. Med. J., **20,** 408.
15. Brain, W. R. (1951): *Diseases of the Nervous System.* London: Oxford University Press.

BOOKS RECIEVED : BOEKE ONTVANG

Stalag Doctor. By I. Schrire. Pp. 209. 13s. 6d. net. London: Allan Wingate (Publishers) Limited. 1956.

Emergencies in Medical Practice. Fifth Edition. Edited by C. Allan Birch, M.D., F.R.C.P. Pp. xii + 684. 155 Illustrations, 12 in colour. 37s. 6d. + 1s. 7d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Progress Report on Birth Control. By Lella Secor Florence. Pp. 260. 21s. net. London: William Heinemann, Medical Books Ltd. 1956.

Lehrbuch der Tropenkrankheiten. By Ernst Georg Nauch. Pp. viii + 432. 125 Illustrations. DM 64. Stuttgart: Georg Thieme Verlag. 1956.

THE USE OF CORTICOSTEROIDS IN COMBINATION WITH ISONICOTINIC ACID HYDRAZIDE IN THE TREATMENT OF ADVANCED BILATERAL PROGRESSIVE CAVITARY PULMONARY TUBERCULOSIS

SECOND REPORT WITH DISCUSSION

A. E. HANDLEY, M.B., B.CH. (RAND)

C.S.I.R. Research Unit in Tuberculosis, King George V Hospital, Durban

Although the picture presented by tuberculosis has been dramatically altered by the advent of drugs such as isonicotinic acid hydrazide (INH) and streptomycin, one of the gravest problems still unsolved is that of long-standing, often partially treated or untreated, pulmonary disease. In these cases, active foci surrounded by fibrosis and avascular tissue remain shielded from medical attack, while the extensive nature of the areas involved in fibrosis and cavitation preclude the possibility of surgical removal. At best they can be maintained *in statu quo* while occupying hospital beds urgently needed for the treatment of acute cases. As out-patients they are dangerous and prolific spreaders of disease.

In the series being reported on, prednisone was added to the routine treatment to determine whether in these cases, the worst of their type, the course of disease could be altered, and also to obtain information which would be of value in the treatment of pulmonary tuberculosis generally.

It was reasoned that prednisone might be of value in the following ways:

(*a*) By reducing inflammatory reaction (exudate, congestion and oedema) and thus allowing better access of medication to tubercle bacilli, better aeration and lung function, and improved drainage of diseased and destroyed tissues. This is of importance also in secondary sepsis.

(*b*) By preventing or retarding further formation of fibrous tissue.

(*c*) By altering the host-reaction to stress (on the assumption that stress, e.g. diet, fatigue and disease, plays an important part in the Bantu type of rapidly-advancing tuberculosis).

(*d*) By improving appetite and general well-being and minimizing unpleasant symptoms.

On 30 June 1956 a preliminary report was published in this *Journal*[1] on a 3 months' trial of prednisone and isonicotinic acid hydrazide in the treatment of a selected group of 23 female Bantu patients with advanced bilateral progressive cavitary pulmonary tuberculosis of poor prognosis. It was reported that 'during the period of the trial no patient died although a fatal outcome would have been anticipated in the majority of cases of this type. All patients exhibited marked clinical improvement. All but one showed satisfactory weight-gains. On independent radiological assessment no patient showed further deterioration and 5 actually showed slight improvement. All sputa remained positive for tubercle.'

The present report covers a total period of 9 months, during which the treatment of this group of 23 patients was as follows:

1. *First period of* 3 *months:* As in the preliminary report[1]—INH (15 mg. daily per kg. body-weight) and prednisone (15 mg. daily).

2. *Second period of* 3 *months:* Without prednisone—INH (15 mg. per kg. daily) and Viomycin (1 g. twice weekly).

3. *Third period of* 3 *months:* On prednisone (30 mg. daily), INH (15 mg. per kg. daily), Dipasic (20 mg. per kg. daily) and streptomycin (1 g. twice weekly).

Prednisone was gradually 'tailed off' at the end of each period.

Throughout the period the patients received extra vitamins, extra proteins and treatment for secondary sepsis.

TABLE OF OBSERVATIONS

	At end of 1st 3-month period (15 mg.)*	*At end of 2nd 3-month period (nil*)*	*At end of 3rd 3-month period (30 mg.*)*
Number of patients showing X-ray improvement	5	9	12
Number of patients showing X-ray deterioration	0	3	4
Average weight-gains (lb.)	20⅛	21⅝	35
	During 1st 3-month period (15 mg.)*	*During 2nd 3-month period (nil*)*	*During 3rd 3-month period (15 mg.*)*
Number of patients having haemoptysis during the period	0	5	1
Number of deaths	0	2	1
Number discharged	0	2	1
Side Effects			
'Moon Face'	3	0	17
Hyperglycaemia	0	0	1
Hirsutism	0	0	1

* daily dosage of prednisone.

All sputa remained positive for tubercle on culture, some being occasionally negative on straight examination.

One month after the end of the trial, all the patients remaining in hospital were ambulant and in good general condition.

DISCUSSION

X-ray Changes

Changes were assessed by independent observations of a panel of 3 senior physicians. By means of numerical evaluation the sums of the monthly variations were

checked against evaluation of changes for the entire period.

During this trial it was suggested that potassium iodide might be as effective as the corticosteroids in aiding the response to INH as shown by X-ray clearing. To settle this point, a series of 18 non-selected tuberculous female Bantu patients were treated with 20 mg. of INH per kg. body-weight, and half of them were given potassium iodide and half of them prednisone. After 1 month X-ray changes were assessed by the independent panel. X-ray clearing in the prednisone group compared to the pot. iod. group was in the ratio of 13 to 8. The average weight-gain in the prednisone group was 9 lb., and in the pot. iod. group 2½ lb. Of the prednisone group, 5 showed good clearing, while of the pot. iod. group 2 showed good clearing.

Deaths

Only 3 patients had died after 9 months, 2 during the second 3-month period while not receiving prednisone. The patient who died while on treatment with prednisone, in the third 3-month period, was well a few hours before death and suddenly had a massive haemoptysis. At post-mortem she was found to have suffered a mechanical rupture of the left pulmonary vein, which was smooth and clean, showing no sign of active tuberculous infiltration, but was passing unsupported across the large cavity which occupied the upper 2/3rds of her left lung. At this, and the other autopsy done, a striking feature was the normal healthy appearance of the myocardium, and the absence of active tuberculous foci in the lungs. (Consent for an autopsy was not obtained in the 3rd case in which the patient died.)

Discharges

Two patients were discharged against medical advice; one of them was doing extremely well, and the other was in poor condition. The third patient discharged is being treated as an out-patient with INH only and is doing very well.

General Condition

As in the preliminary report,[1] patients receiving prednisone exhibited striking improvement in mood, sleep and appetite. During the second 3-month period (while not receiving prednisone) there was a general tendency toward deterioration of general condition at the end of the 3rd month. This, however, was not sudden or alarming and there was still a great improvement as compared with the condition before treatment with prednisone was begun.

Weight Gains

Although a certain proportion of the increase in weight was undoubtedly due to sodium and water retention, it was felt that the general beneficial effect of this hormone and the suppression of the disease were inducive of a healthy increase in weight. Prednisone exerts a catabolic or anti-anabolic effect in protein metabolism, thereby suppressing the synthesis of protein, but to what extent this affects normal tissue is not known. It certainly is most advisable to make extra-readily assimilable protein available to patients on prednisone therapy. During these trials 'Davein' (protein hydrolysate with vitamin B-complex) has been given, and it was observed that, on high dosage of corticosteroids 'moon face' became more marked when the Davein was withheld and was largely reversed when it was given again.

Haemoptysis

An unexpected observation was the marked diminution in the incidence of haemoptysis. Small doses of prednisone in combination with vitamins C and K and antibiotics have, in fact, been successfully used in other cases of bleeding. It is suggested that this is due to reduction in capilliary fragility and inflammation.

Carbohydrate Metabolism

All patients exhibited mild glycosuria after 10-14 days on 30 mg. of prednisone. One patient suddenly (within 24 hours) developed symptoms and signs of hyperglycaemia (fasting blood-sugar 428 mg.%). The blood-sugar curve was typical of diabetes mellitus. The patient suffered a violent reaction to only 10 units of soluble insulin, given as a test dose. Prednisone was stopped, and after 19 days the sugar content of blood and urine was normal. Prednisone was then renewed (15 mg. daily) without further untoward effects.

Hirsutism

One patient on 30 mg. of prednisone developed mild hirsutism, which has not disappeared 1 month after cessation of prednisone treatment.

Dosage

It will be noticed that a daily dosage of 30 mg. of prednisone produced more side-effects but no proportional clinical or radiological improvement. The optimum dosage for the average adult appears to be 15 mg. daily.

SUMMARY AND CONCLUSION

A 9 month's trial is described of the use of prednisone in combination with isonicotinic acid hydrazide in the treatment of a series of 23 patients (Bantu females) with advanced bilateral progressive cavitary pulmonary tuberculosis of poor prognosis.

The probable mode of action, dosage and side-effects are discussed.

Bearing in mind that these patients were all suffering from very advanced pulmonary tuberculosis it can be stated that:

1. Prednisone may safely be given to tuberculous patients with beneficial effect, provided that the tuberculous disease as well as secondary infections are adequately controlled by antibiotic therapy and chemotherapy.

2. Prednisone appears to speed up initial response to INH therapy in non-selected cases of pulmonary tuberculosis, and to be more efficacious than potassium iodide for this purpose.

I should like to thank Dr. B. A. Dormer, Medical Superintendent of the King George V Hospital, Durban, for his continued guidance and help. We are indebted to Messrs. Scherag (Pty.) Ltd. for supplies of prednisone (Meticorten) used during this trial.

1. Handley, A. E. (1956): S. Afr. Med. J., **30**, 605.

CREATINE EXCRETION IN DIABETES MELLITUS

W. M. POLITZER AND T. SCHNEIDER

South African Institute for Medical Research and the General Hospital, Johannesburg

Dupuytren's contracture is a common condition in diabetes mellitus. One of the authors has described 120 of these cases among 381 diabetics in the older age-group.[1]

Steinberg[2] has stated that in Dupuytren's contracture the creatine excretion in the urine is increased and suggests that both in this condition and in fibrositis there is an abnormality in tissue metabolism. He found that the total amount of creatine excreted per 24 hours in cases of primary fibrositis was 264-918 mg. (in this group he included Dupuytren's contracture). In 12 out of 15 cases it was over 300 mg. per 24 hours.

In view of this suggestion and the frequency of Dupuytren's contracture in diabetes, it was decided to investigate the creatine excretion in a series of diabetic patients.

Method

The investigation comprised 74 adult European patients (50 females and 24 males) attending the Diabetic Clinic of the Johannesburg General Hospital, of whom 39 (29 females and 10 males) had Dupuytren's contracture. Their ages varied from 45 to 83 years (average 62½ years). A control group of 50 patients of a similar average age and not suffering from either diabetes or Dupuytren's contracture or any other condition known to affect creatine metabolism were similarly investigated.

The total creatine was determined on 24-hour specimens of urine collected from each patient. The method used was that of Peters and Van Slyke.[3] As shown in Table I the total diabetic group revealed a significant increase in creatine excretion; those with Dupuytren's contracture also showed a similar increase. In the control group only one female exhibited a 24-hour creatine excretion of over 300 mg. whilst in the diabetic groups 2 males and 11 females had this high excretion rate. Of the latter 2 males and 3 females showed Dupuytren's contracture.

TABLE I. CREATINE EXCRETION IN MG. PER 24 HOURS

	Females	*Males*
Diabetic group	185(18-858)	164(20-782)
Diabetics with Dupuytren's contracture	164(14-858)	278(47-782)
Controls	99(20-420)	87(18-213)

DISCUSSION

It is well known that in conditions with muscle degeneration or destruction an increase in creatine excretion occurs.[4] Thus, in muscular dystrophy some of the highest creatine excretion figures are obtained. Steinberg has drawn attention to the fact that in primary fibrositis a marked creatinuria occurs; and having placed Dupuytren's contracture in the same group of cases he infers that here too creatine values in the urine are increased.

While this may be true in non-diabetic subjects it is interesting to note that our own figures among female diabetics reveal no significant difference between the group with and the group without Dupuytren's contracture. On the other hand, amongst our male diabetics with Dupuytren's contracture there is a marked rise in the creatine excretion compared with the total diabetic group and the control group. While the group is small in numbers some significance must be attached to the differences noted.

The main observation which emerges from this investigation is that creatinuria was found to be very common in the diabetic population examined. Of 74 patients 13 (18%) had a creatinuria of over 300 mg. per day compared with the control group with 1 out of 50 (2%). It would therefore appear that creatinuria is related to the diabetic state. Age itself is not of any significance as demonstrated by the control group. Neither does Dupuytren's contracture in the diabetic female appear to influence creatinuria unduly. In the male, however, figures of 782, 736, 260, 246, 228, 135, 135, 120, 86 and 47 mg. were obtained. In view of the fact that creatine excretion in the healthy male occurs irregularly and is usually only half or less than half that in the female some significance must be attached to the figures obtained. The possibility therefore that this may be related to Dupuytren's contracture as suggested by Steinberg cannot be excluded; a large series of cases would have to be analysed before a definite opinion could be given.

It has been suggested in the past that diabetes causes creatinuria through the break-down of muscle tissue[4] following upon impaired utilization of glucose. No direct relationship was found between the degree of stabilization of diabetes and the amount of creatine excreted in the urine; for instance patient G.M.S., who was under good diabetic control, had a creatine excretion of 858 mg. whilst Sister G., a poorly controlled, brittle diabetic, had a creatine excretion of 58 mg. per 24 hours. Neither was the creatine excretion in the urine influenced by the duration of diabetes. Thus B.S., a diabetic of 18 years' standing, had a creatinuria of 18 mg. per 24 hours, while after 3 years G.M.S. excreted 858 mg. creatine.

Our observations therefore lead us to believe that in the older age-groups of diabetics creatinuria is common, both in those with and without Dupuytren's contracture. The latter condition was not found to influence the creatinuria to any extent in the female cases examined although in the male group there was evidence to suggest that Steinberg's contention of the association of Dupuytren's contracture with creatinuria might be supported.

SUMMARY

1. Creatine excretion was determined in a group of 74 European diabetics (50 females and 24 males) and 50 control cases.

2. Thirty-nine diabetics (29 females and 10 males) had Dupuytren's contracture.

3. Creatinuria was greater in the diabetic group.

4. There was no significant difference between the creatinuria in the female total diabetic group and in those with diabetes plus Dupuytren's contracture, but there was a marked difference between the males of the two groups.

5. The significance of the results is discussed.

We wish to thank Mr. V. J. Noble for his technical assistance.

REFERENCES

1. Schneider, T. (1953): S. Afr. Med. J., **27**, 466.
2. Steinberg, C. Le R. (1946): Med. Clin. N. Amer., **30**, 221.
3. Peters, P. and Van Slyke, D. (1932): *Quantitative Clinical Chemistry*, vol. 2, p. 602. London: Baillière, Tindall and Cox.
4. Wright, S. (1952): *Applied Physiology*, p. 894. London: Oxford University Press.

ASSOCIATION NEWS: VERENIGINGNUUS

MEDIESE BESKERMING

Op die jongste vergadering van die Federale Raad is dit ooreengekom dat die Mediese Vereniging van Suid-Afrika 'n ooreenkoms met die Medical Protection Society van Londen sou sluit, waardeur die Genootskap 'n Suid-Afrikaanse Tak in oorleg met die Vereniging sou stig.

Vir baie jare is beskerming aan ons lede verskaf volgens 'n ooreenkoms met die Atlas Assuransie Maatskappy, wat waardevolle diens aan die mediese professie in ons land gelewer het deur die dekking wat hulle voorsien. Hegte vriendskapsbande het bestaan, en bestaan nog tussen die maatskappy en die Vereniging, en die ooreenkoms wat nou met die Medical Protection Society aangegaan is, verskaf 'n alternatiewe diens aan lede maar vervang nie die diens wat die Atlas Maatskappy gedurende al hierdie jare voorsien het nie.

'n Memorandum wat die dienste van die Medical Protection Society duidelik maak, is vir oorweging aan al die lede van die Vereniging gepos, en dit word aan die lid oorgelaat om te besluit watter vorm van beskerming hy verkies. Een ding staan egter vas, naamlik dat elke dokter op een of ander wyse beskerm moet wees. Versuim om hierdie voorsiening te tref, asook nalatigheid om toe te sien dat die beskerming, met betrekking tot die bedrag van indemniteit, doeltreffend is, is dwaasheid.

Dit is gevind dat die vorige laer perk van £1,000 nie meer redelike en genoegsame beskerming bied nie, en dit is nou tot £2,000 verhoog. Sommige soorte van praktyke het beskerming teen baie groter eise nodig.

Die vorms wat gesirkuleer is, sluit 'n aansoekvorm in, en die aanvangsdatum vir dekking deur die Medical Protection Society dateer van die datum waarop die voltooide vorm by die kantoor van die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, ontvang word. Lede wat miskien besluit om die beskerming wat hulle by een of ander maatskappy of genootskap het, te verander, moet aandui wanneer die volgende hernuwingspremie normaalweg betaalbaar is.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Ef-Cortelan Nasal Spray. This product is an isotonic aqueous solution containing: Hydrocortisone (alcohol) 0·02%, naphazoline nitrate 0·025%.

Hydrocortisone has been shown to be effective for the relief of inflammatory conditions of the nasal passages when applied to the mucous membranes of the nose in very dilute solutions. Local application avoids the possibility of systemic effects and the low concentration is unlikely to cause irritation. The presence of naphazoline, a decongestant vasoconstrictor, assists the hydrocortisone to exert a maximum effect.

Ef-Cortelan Nasal Spray is recommended for the commonly occurring inflammatory allergic conditions of the nose, such as hay-fever and vasomotor rhinitis. It is also effective in reducing the inflammation in sinusitis and helps to prevent the recurrence of nasal polypi.

The length of treatment varies with the condition and the response obtained, but unnecessarily prolonged treatment should be discouraged. Where acute infection is present this may require appropriate treatment concurrently.

Manufactured by Glaxo Laboratories (S.A.) (Pty.) Ltd., P.O. Box 21, Wadeville, Transvaal. Packed in 15 c.c. plastic squeeze-bottles.

* * *

Neobacrin Ointment. This product contains 5 mg. of neomycin sulphate and 500 units of zinc bacitracin per gram, in a bland paraffin base. Both these antibiotics are bactericidal. They are non-irritant and have an extremely low sensitizing potential. No systemic effects from Neobacrin have been observed, even when applied to large body-surfaces for prolonged periods.

Neobacrin is recommended for the treatment of pyogenic skin conditions such as impetigo, sycosis barbae, furunculosis, and secondarily infected wounds and burns. Superficial infections of the eye such as blepharitis, conjunctivitis and styes also respond well to Neobacrin.

Manufactured by Glaxo Laboratories (S.A.) (Pty.) Ltd., P.O. Box Wadeville, Transvaal. Packed in 3·5 gram long-nozzle tubes.

REVIEWS OF BOOKS : BOEKRESENSIES

AIDS TO DISPENSING

Aids to Dispensing. Fifth Edition. Revised by G. M. Watson, B.Pharm., F.P.S. Pp. vii + 167, with 7 Illustrations. 7s. 6d. net. London: Baillière, Tindall and Cox Ltd. 1956.

Contents: Preface. I. Introduction. II. Powders. III. Cachets and Capsules. IV. Percentage Solutions. V. Mixtures. VI. Emulsions. VII. Incompatibles. VIII. Pills. IX. Pill Coating. X. Tablets. XI. Pastilles and Lozenges. XII. Effervescing Granules. XIII. Lotions. XIV. Other External Applications. XV. Preparations for use in the Eyes. XVI. Ointments. XVII. Suppositories. XVIII. Preparation of Isotonic Solutions. XIX. Preparation of Sterile Products. XXI Antibiotics. Index.

The 'Aids' series has always been popular with students and 'Aids to Dispensing' has now reached its 5th edition, having first appeared in 1928. Pharmaceutical practice, like all forms of practice, has changed considerably since those days and in his revision the author has sought to bring the book completely up to date in regard to both knowledge and methods. Certain

new methods of manufacture, such as the dry coating of tablets and the rotary-die method of preparing gelatine capsules, have been described briefly and the chapter on antibiotics has been rewritten, setting out the responsibilities of those who handle these preparations in a realistic way. Both the imperial and metric systems of calculating quantities have been used.

Although it is students of pharmacy who will be primarily interested, this work will also be of value to medical students studying pharmacology.

A.H.T.

NEUROLOGICAL NURSING

Neurological Nursing. A Practical Guide. By John Marshall, M.D., M.R.C.P., M.R.C.P.Ed., D.P.M. Pp. vii + 166. Figures 83. 18s. 6d. Oxford: Blackwell Scientific Publications. 1956.

Contents: 1. Introduction to the Neurological Unit. 2. Anatomy and Physiology. 3. The admission and examination of the patient. 4. The care of the skin and the prevention and treatment of pressure sores. 5. The management of neuroligical disorders of the bladder. 6. Nursing the unconscious patient. 7. Passive movements and rehabilitation. 8. The management of epilepsy. 9. Special neurological investigations. 10. The causes and management of respiratory difficulties. 11. Preoperative and post-operative nursing care. 12. The psychological approach to the patient with neurological disease. 13. Social care in neurological disease.

It is only comparatively recently that the imperative need for the skilled nursing of neurological cases has become obvious, and, until this excellent book of Dr. Marshall's appeared simultaneously in England and America and Canada last May, no suitable book existed on this subject.

In his short foreword to Dr. Marshall's book, Dr. W. Ritchie Russell, one of the most eminent neurologists of our time, says: 'Neurologists are often so busy that they have little time to do anything else but to diagnose and to leave the treatment to others to do. The present-day neurologist however, realizes more than ever before the need for organizing treatment for his patients. . . . Clearly there is a need here for postgraduate nursing training in neurology, and Dr. Marshall has made an important contribution to the problems involved by writing this excellent account of the neurologically trained nurse'.

'Neurological Nursing', as its author explains from the outset, has been specificially written for nurses, doctors, physiotherapists and others concerned with the practical care of patients suffering from neurological disease. It is in no way intended as a medical handbook of any kind; musch less as a text-book on neurology. It is more a guide-book for those into whose hands the care and comfort—sometimes the very life—of the patients are entrusted.

All the specific nursing procedures that may be encountered in the course of neurological nursing are carefully partitioned off into chapters, each clearly headed, and there is no unnecessary verbiage to confuse the reader or leave him in any doubt as to the author's meaning. The delicate and extraordinarily meticulous care imperative in post-operative care after brain surgery is gone into in great detail, as is the nursing of conditions such as epilepsy, concussion and the state of unconsciousness following upon many surgical and medical conditions.

I find the chapter on the care of the skin, which is after all one of the first things upon which stress is laid in the elementary training of every nurse, unnecessarily protracted, but that may be due to my own unfamiliarity with many of the latest methods and needs in neurological nursing. But Chapter 10, on the management of respiratory difficulties, is admirable both in scholarship and lucidity and of great value not only to nurses but to doctors and anaesthetists as well.

In 'Neurological Nursing' we have a book which covers a wide field of practical nursing in an authoritative yet unassuming fashion. It avoids over-elaboration but omits no essential. By any standards a most excellent book, and one to be highly recommended.

L.B.

DISEASES OF THE CHEST

Diseases of the Chest. By H. Corwin Hinshaw, M.D., Ph.D. and L. Henry Garland, M.B., B.Ch. Pp. x + 727 + 634 Illustrations on 277 figures. $15.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: 1. Diagnostic Procedures. (Clinical History and Evaluation of Complaints). 2. Diagnostic Procedures (Physical Examination and Bronchoscopy). 3. Diagnostic Procedures (Laboratory Studies). 4. Diagnostic Procedures (Radiologic Examination of the Thorax). 5. Segmental Anatomy of the Tracheobronchial Tree and Lungs. 6. Measurement of Pulmonary Function By Roger H. L. Wilson. 7. The Bacterial Pneumonias. 8. Pneumonias of Viral and Rickettsial Etiology—Summary of Roentgenography in Pneumonia. 9. Pulmonary Inflammation and Fibrosis due to Physical, Chemical and Obscure Causes. 10. Pulmonary Abscess. 11. Inflamatory and Suppurative Diseases of the Bronchi. 12. Postoperative Pulmonary Atelectasis. 13. Foreign Bodies in the Larynx and Tracheobronchial Tree By Walter E. Heck, M.D. 14. Thoracic Injuries. 15. Diseases of the Diaphragm. 16. Bronchial Asthma and Related Conditions. 17. Pulmonary Emphysema. 18. Pulmonary Cysts, Bullae and Blebs—Spontaneous Pneumothorax. 19. Mediastinal Diseases. 20. Bronchogenic Carcinoma. 21. Bronchial Adenomas and Some Other Intrathoracic Tumors. 22. Intrathoracic Metastases. 23. Pulmonary Congestion and Edema. 24. Pulmonary Embolism. 25. Congenital Anomalies. 26. Tuberculosis (Bacteriology and Pathogenesis). 27. Pulmonary Tuberculosis (Diagnostic Procedures and Classification). 28. Treatment of Tuberculosis (Rest and General Management). 29. Treatment of Tuberculosis (Specific Antibacterial Drugs). 30. Treatment of Pulmonary Tuberculosis (Collapse Therapy). 31. Treatment of Pulmonary Tuberculosis (Pulmonary Resection). 32. Medical Aspects of Tuberculosis Control. 33. Diseases of the Pleura. 34. Sarcoidosis. 35. Pulmonary and Associated Changes in the Collagen Diseases. 36. Coccidioidomycosis By William A. Winn, M.D. 37. Actinomycosis, Nocardiosis and Blastomycosis. 38. Histoplasmosis and Some Other Pulmonary Mycoses. 39. Parasitic and Other Tropical Pulmonary Diseases. 40. Pulmonary Diseases of Occupational Origin. Index.

This is a very comprehensive, up-to-date text-book, suitable for both undergraduate and postgraduate students and practitioners. Radiology plays such a large part in the diagnosis of chest diseases today, that the joint authorship by a professor of clinical medicine and a professor of clinical radiology would seem a wise choice. The text is brief and yet extremely comprehensive and the numerous roentgenograms are excellently reproduced. There is a full chapter by a separate author on the measurement of pulmonary function—a subject of increasing importance today. The writer could find no information about the subject of fat embolism—the only small point of criticism which can be offered.

It is a welcome change to find references to British literature freely quoted in an American text-book of this sort, and all round it is a most well-balanced and up-to-date review of the subject. One has little hesitation in giving it the strongest recommendation.

M.J.B.

CURRENT THERAPY

Current Therapy—1956. Latest Approved Methods of Treatment for the Practising Physician. Edited by Howard F. Conn, M.D. Pp. xxx + 632. $11.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: 1. The Infectious Diseases. 2. Diseases of the Respiratory System. 3. Diseases of the Cardiovascular System. 4. Diseases of the Blood and Spleen. 5. Diseases of the Digestive System. 6. Disorders of Metabolism and Nutrition. 7. Diseases of the Endocrine System. 8. Diseases of the Urogenital Tract. 9. The Venereal Diseases. 10. The Allergic Diseases. 11. Diseases of the Skin. 12. Diseases of the Nervous System. 13. Diseases of the Locomotor System. 14. Obstetric and Gynecologic Conditions. 15. Diseases Due to Physical and Chemical Agents. 16. Appendices and Indices.

This volume represents the eighth edition of an annual series, and is beautifully presented to the practising physician, with an excellent index of subject, covering very adequately the whole field of modern therapy, with current views and modifications. There are upwards of 300 contributing authors, all but two of them of the North American Continent, and all considered to be recognized authorities. The two 'outsiders' are Dr. Smirk of Dunedin, New Zealand, who writes on the treatment of Hypertension, and Dr. Sheehan of Liverpool, whose subject is Hypopituitarism.

The treatments and methods described are thoroughly up to date. With such a multiplicity of authorship there is of necessity a wide variation in style and quality of the individual articles.

The volume is recommended for use and reference by the busy general practitioner and specialist. If this continues to be an annual edition, each of which is virtually a new book rather than a revision of the previous edition, then one wonders whether a 'loose-leaf' edition would not be more acceptible, more especially as it is an expensive book to replace each year.

A.L.

THE PARKINSONIAN PATIENT

Extrapyramidaal Syndroom of Situatief Gedrag. Door dr. A. C. Lit. Blz. 251. f 12.50. Amsterdam: Noord-Hollandsche Uitgevers Maatschappij. 1956.

Inhoud: Inleiding. *Het Syndroom van Parkinson in de Literatuur*. I. De theoreën over de aetologie. II. De motorische verschijnselen. III. De psychopathologische verschijnselen. IV. Het post-encephalitisch Parkinsonisme. V. De psychoso-

matische opvattingen. *Het onderzoek.* VI. De patiënten. VII. Het psychiatrisch onderzoek. VIII. De biografische anamnese. IX. Samenvatting van her psychiatrisch en biografisch onderzoek. X. Het psychologisch onderzoek. XI. De motorische verschijnselen. XII. Enkele prognotische en therapeutische conclusies. Samenvatting. Summary. Zusammenfassung. Sommaire. Literatuurlijst.

This is a phenomenological study, i.e. in it the author discusses not so much the Parkinson syndrome as the parkinsonian patient.

Although the author accepts the anatomical findings in cases of paralysis agitans and so-called postencephalitic parkinsonism, he feels that they cannot explain the clinical picture sufficiently. Lesions in the globus pallidus, the ansa lenticularis and the substantia nigra are, moreover, found *postmortem* without there having been a typical Parkinson syndrome during life.

The author thereafore attempts to approach the problem from a psychological angle. He stresses the premorbid temperament, which closely resembles Janet's psychasthenia and which is invariably found in cases of paralysis agitans and to a lesser degree in parkinsonism. His psychological investigations (Rorschach test and Wechsel-Bellevue method) confirm these psychiatric and biographic findings. For these people 'the aspect of the world implies a threat rather than an obvious invitation. They have a tendency to distantiation and suppression of emotional life'. The majority of patients, nevertheless, had succeeded in establishing a satisfactory if formal adjustment. When, however, endogenous and exogenous disturbing influences supervene, this type of person gives up the struggle and 'distantiates' himself further into the akinesia, rigidity and tremor of the Parkinson syndrome. The author considers that the disease is more readily understood as a reaction of a temperament to a situation 'it is inextricably correlated with changes in the person—world relationship') than explained by an organic lesion.

It is impossible to do justice to a study of this kind in a short review. While admitting that it may be useful to view an organic nervous disease also from a psychological angle, one feels that the author is in danger of ignoring the fact that the organic brain changes remain a condition *sine qua non* for the causation of the Parkinson syndrome.

The author is on safer ground when he stresses the necessity for psychotherapy and physiotherapy. This is too often neglected in the treatment of the disease, which in many instances is only combated by prescribing the usual older and newer neuroplegics. That this warning is not new, is well known to all neurologists who remember the era of the Bulgarian root and the special clinics for parkinsonians, which were *en vogue* after the first world war.

As a thoughtful and often original phenomenological study however, the book has considerable value.

F.H.K.

PHYSICAL DIAGNOSIS

Physical Diagnosis. Fifth Edition. By Ralph H. Major, M.D. and Mahlon H. Delp, M.D. Pp. xi + 358. 536 Figures. $7.00. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: 1. Introduction. 2. History Taking and Recording. 3. Pain. 4. General Inspection. 5. Examination of the Head and Neck. 6. Inspection, Palpation and percussion of the chest. 7. Auscultation of the Lungs. 8. The Physical Findings in Diseases of the Lungs. 9. Inspection, Palpation and percussion of the Heart. 10. Auscultation of the Heart. 11. The Pulse. 12. Blood Pressure. 13. Physical Findings in Cardiovascular diseases. 14. Abdomen and Genitalia. 15. The Extremities. 16. Examination of the Nervous System. Index.

It is nearly 20 years ago that this book first appeared and it has served its purpose well. At that time the author made it plain that he laid great stress on the appreciation of the meaning of the physical signs in illness and for that reason he avoided reference to X-ray, pathological and other forms of diagnostic aids. He makes frequent use of the descriptions of the 'old masters' and mentions how impressed he has always been by the words of Sir William Osler, 'and when you can, read the original descriptions of the masters who, with crude methods of study, saw so clearly'.

In these days when so many aids to diagnosis are known and so frequently used, often at great cost to the patient, it is well that a book like this should be in its 5th edition.

The original author welcomes Dr. Mahlon Delp as a co-author and pays tribute to his help in the past. This new edition contains a number of additions to both the text and the illustrations and many parts have been re-written. A noteworthy feature is the transfer of the chapter on history—taken from the end of the book to a prominent place at the beginning.

A.T.

A TEXT-BOOK OF MEDICINE

The Principles and Practice of Medicine. A Text-book for Students and Doctors. Third Edition. By Sir Stanley Davidson, B.A. (Cantab.), M.D., P.R.C.P.Ed., F.R.C.P. (Lond.), M.D. (Oslo). Pp. viii + 1,076. 73 Figures. VII Plates. 35s. net. Postage 1s. 9d. Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1956.

Contents: Infection and Disease. Infectious Diseases. Chemotherapy. Diseases of the Respiratory System. Nutritional Disorders. The Chronic Rheumatic Diseases. Tropical Diseases and Helminthic Infestation. Disorders of the Blood and Blood-Forming Organs. Diseases of the Endocrine System. Diseases of the Kidney and Urinary System. Disturbances in Water and Electrolyte Balance and Acid-Base Equilibrium. Diseases of the Digestive System. Diseases of the Liver and Biliary Tract. Diseases of the Pancreas. Diseases of the Nervous System. The Psychoneuroses. Appendix A—Diet Sheets. Appendix B—Tables of Standard Body Weights. Index. Tables of Weights and Measures and Exchanges.

This book was first published in 1952 and within the short space of 3½ years the 1st and 2nd editions and two large reprints have been sold. It would seem obvious that this phenomenal demand has been the result of its approval not only by students and their teachers but by doctors generally.

The contributors are members of the staff of the Department of Medicine of the University of Edinburgh and it is clear that there has been close cooperation between them. This had led to a balanced style and composition and the presentation of the material is excellent. In addition, the whole work shows the diversity and breath of knowledge and experience which must result from the team-work of outstanding physicians.

In this 3rd edition the section on neurology has been re-written and the bringing up to date of the other sections has caused an increase in the size of the book by some 38 pages. The illustrations are clear and sufficient. It is a good book, which undoubtedly deserves the success it has achieved.

A.T.

CLINICAL ABSTRACTS FROM J.A.M.A.

J.A.M.A. Clinical Abstracts of Diagnosis and Treatment. Published with the Approval of the Board of Trustees, American Medical Association. Pp. vii + 661. $5.50. New York and London: Intercontinental Medical Book Corporation with Grune & Stratton, Inc. 1956.

Contents: Preface. Digestive System. Respiratory System. Cardiovascular System. Urinary System. Reproductive System: Pregnancy, Breast, Tubes, Ovaries, Vagina, and Uterus, Testes. Endocrine System. Bones, Joints, and Connective Tissue. Blood and Lymphatic System. Skin. Nervous System. Eye, Ear, Nose, and Throat. Metabolism. Poisonings. Infections. Therapeutics: Anesthesia, Shock, Fluid Therapy, Cancer, New Drugs, Miscellaneous. Diagnostic Technics. Index of Subjects.

The need for practitioners to keep up to date has stimulated the production of numerous books. We have annual volumes of all kinds, some general, some specialized. This one is said to rest on a broad basis, providing specialized knowledge for the general practitioner and keeping the specialist abreast of developments in fields other than his own. By the use of this type of book the busy doctor can take his 'refresher course' in comfort and in a relatively painless fashion!

Books in this series have been published twice before. It is a selected group of 'medical literature abstracts' which have appeared in the Journal of the American Medical Association. Those who read medical journals will have seen many of these papers in their original form. The appeal of this volume will therefore be to those who lack either the time or inclination for extensive reading of current medical literature.

There is a further restriction. This volume deals almost entirely with diagnosis and treatment. This is said to establish both economy of words and reading time but not of ideas! But it must restrict its usefulness.

The abstracts are well selected and most of the important papers of the current year will be found in its pages. They are grouped under systems which make for easy reference. There is something for everyone but not a great deal for anyone with a particular interest. Those who like this kind of volume will find it as good as most of its kind but not as good as the best. Those who read it are bound to pick up useful 'tips' but no profound advance in knowledge should be expected by the perusal of its pages.

C.M.

THERAPY OF DISEASES OF THE HEART AND CIRCULATION

Klinische Pharmakologie der Herz- und Kreislaufkrankheiten als Grundlage einer individuellen Therapie. By Hans Seel, M.D. Pp. 268. DM 19.60. Hippocrates-Verlag, Stuttgart, Germany. 1956.

Contents: I. Clinical pharmacology as a basis for individual therapy. The doctor. The patient. The Drug. II. Pathologic-physiological points of view for the treatment of diseases of the heart and circulation. III. Drugs for the treatment of diseases of the heart and circulation. A. Drugs with a specific action on the heart: (1) Strophanthus-Digitalis group. (2) Glycosides of the II order—digitaloids. (3) Non-glucosidal cardiacs. (4) Organpreparations (ATP, heart extracts, liver extracts). (5) Hormones. (6) Alkaloids. B. Drugs with a specific extra-cardiac action: (1) Purin group. (2) Drugs which stimulate the central nervous system. (3) Sympathomimetics. (4) Sympatholytics and adrenolytics: (*a*) Vasodilatory sympathomimetics: (*b*) Nicotinic acid and its derivatives. (5) Ganglion-blocking agents. (6) Parasympatholytics. (7) Spasmolytics. (8) Nitrite group. C. Drugs used in the treatment of diseases of the peripheral bloodvessels: (1) Diseases of the arterial system. (2) Diseases of the venous system. IV. Clinical-pharmacological guides and critique of the therapy.

Before Seel took up the chair in Clinical Pharmacology at the University of Berlin, he was Director of the Research Institute for Clinical Pharmacology in Hamburg-Eppendorf. The nature of Seel's researches through many years and his extensive experience in medical practice certainly render him one of the most capable authors of a book on clinical pharmacology. From the summary of the contents given above, it is obvious that the author has covered a very wide field which is of the utmost importance not only to specialists in diseases of the heart and circulatory system, but also to research workers and general practitioners. In the space available the subject-matter of the book cannot be reviewed in detail. An important point which deserves to be mentioned, is Seel's conclusion, as a result of his practical experience as a heart specialist, that different types of heart disease require different doses of digitalis. The author discusses the use of drugs which are either not mentioned in books on pharmacology or are referred to only cursorily. Also the use of spartein and iodine in arteriosclerosis therapy receive detailed attention. The discussion of the drugs in the light of the nature of their actions on the system, the extensive old and new literature quoted, and the effects of nutrition, circumstances and other sociological conditions on diseases of the heart and circulation, add materially to the value of this important publication.

D.G.S.

PSYCHIATRY FOR STUDENTS AND NURSES

A Practical Handbook of Psychiatry for Students and Nurses. New Third Edition. By Louis Minski, M.D., F.R.C.P., D.P.M. Pp. 144. 7s. 6d. net. London: William Heinemann—Medical Books—Ltd. 1956.

Contents: Preface. Introduction. I. Development of the Individual. II. Child Psychiatry. III. Aetiology of Mental Illness. IV. General Symptomatology. V. Affective Reaction Types. VI. Schizophrenic Reaction Types. VII. Organic Reaction Types. VIII. Organic Reaction Types (continued). IX. Organic Reaction Types (continued). X. Psychononeuroses. XI. Nursing and General Management. XII. Specialised Forms of Treatment (Insulin). XIII. Specialized Forms of Treatment (continued). (Convulsion Therapy, Electronarcosis, Carbon Dioxide Inhalation Therapy, Continuous Narcosis, Prefrontal Leucotomy and Malarial Treatment of General Paralysis) Largactil, Serpasil. XIV. Psychotherapy. XV. Occupational Therapy and Rehabilitation. XVI. Legal Aspects of Psychiatry. Index.

This little handbook gives the essentials of a knowledge of psychiatry in a short and concentrated form. In this third edition the author has revised it so as to include the changes and advances in treatment which have been made in this important field of practice in recent years.

Its appeal will be mainly to students and nurses who require an authoritative survey when preparing for examinations, but it has an interest also for general practitioners who may not be able to spare the time to read longer works and yet wish to keep up to date in this subject.

A.T.

CORRESPONDENCE : BRIEWERUBRIEK

STATUS THYMICO-LYMPHATICUS

Letter under date 11 *September* 1956 *addressed to Dr. O. V. S. Kok, Pretoria, by Dr. W. N. Kemp,* 2414 *Main Street, Vancouver* 10, *B.C.*

Thank you for your courteous letter of 31 August last and for the reprint of your valued paper[1] on Status Thymico-lymphaticus which I have read with great interest, particularly the description of your animal experimentation; which experimentation would seem to disprove my hypothesis of the adrenal-thyroid etiology of status lymphaticus in humans.

May I here call your attention to the early experimental studies of Wisloki and Crowe (Bull. Johns Hopkins Hospital, October 1914), which studies were directed at the elucidation of the function of the adrenal cortex and only incidentally threw some light on the human syndrome of status lymphaticus.

Wisloki and Crowe removed 7/8ths of dogs' adrenal cortices. Dogs which survived the operation enjoyed normal functions in all respects except that they had lower resistance to upper respiratory infections than their control litter-mates.

One of these animals was accidentally killed and the autopsy report reads exactly like that of one of the hundreds of children who have died in Vancouver *without anaesthesia or surgery* in which the pathologists' diagnosis has invariably been 'status lymphaticus'. This is a report of a dog, 18 months of age, who survived partial adrenalectomy for 4½ months before being accidentally killed. During this period he gained 5½ pounds and his skeletal growth and other development and function was normal. The post-mortem findings were as follows:

Thymus gland: large and vascular, measuring 14×6×2·5 cm. On section seen to be packed with lymphocytes with numerous large Hassall's bodies.

Lymph glands: the mesenteric and mediastinal lymphatic glands were remarkably enlarged.

Peyer's patches: the lymphoid follicles in the lower part of the small intestine were large and prominent.

Tonsils: normally there are no tonsils visible in a dog but the present subject had definitely large tonsils.

It has been noted by Banting *et al.* (Amer. J. Physiol., 77, 100, 1926) that following complete adrenalectomy and before the animal's death in approximately one week there occurred marked enlargement of the thymus gland and of all lymphatic glands. Hartman in a personal communication in 1931 informed me (*a*) that adrenalectomized cats maintained in apparent normal health by cortical extract readily die under ether anaesthesia; and (*b*) that adrenalectomized but treated rats can ill withstand temperatures that do not disturb litter-mate controls.

The work of Wisloki and Crowe clearly indicates that a physical condition exactly simulating the post-mortem findings in children dying (without anaesthesia) because of 'status lymphaticus' can be induced in dogs by the removal of 7/8ths of their adrenal cortices. It would seem to be highly probable that if one were to excise 7/8ths of a dog's adrenal and then, after complete recovery from this surgery, were to deprive the dogs of dietary iodine that a true *experimental status lymphaticus* would be induced. Then, such a dog, or dogs, could be studied in reference to ability to withstand the stress of anaesthesia and, possibly, surgery.

Whether one can induce experimental status lymphaticus in young rats I do not know. If this can be done after the method of Wisloki and Crowe, these animals would be suitable for further experimentation but *only after they had been placed on an iodine-free diet.* However, I doubt if rats can be used to reproduce the same post-mortem findings as the Wisloki-Crowe dogs.

I sincerely hope that you will continue your experimental interest in this subject, which has bedevilled and bewildered the medical profession since 1614, when Felix Plater reported the first cases in Switzerland, long notorious as a goitre area.

I am enclosing a copy of this letter in the hope that you will persuade the Editor of the *South African Medical Journal* to publish it as a follow-up of your own worthy paper as presented at the South African Medical Congress in October 1955.

1. Kok, O. V. S. (1956): S. Afr. Med. J., **30**, 653.

South African Medical Journal

Organ of the Medical Association of South Africa

Incorporating the *South African Medical Record* and the *Medical Journal of South Africa*

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Cape Town, 29 December 1956
Weekly 2s. 6d.

Vol. 30 No. 52

Kaapstad, 29 Desember 1956
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IN THIS ISSUE — IN HIERDIE UITGAWE

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Applications must be addressed to the Medical Superintendent, Dr. Van Niekerk Hospital, Springbok and are to reach his office not later than 31 December 1956.

C. J. N. Basson
Medical Superintendent

DR. VAN NIEKERK HOSPITAAL, SPRINGBOK
VAKATURES VIR ERE-MEDIESE PERSONEEL

Aansoeke word ingewag van geregistreerde plaaslike Mediese Geneeshere vir aanstelling op die Dr. Van Niekerk Hospitaal se ere-mediese personeel.

Aanstellings, diensvoorwaardes en besoldiging aan bogenoemde poste verbonde, sal onderworpe wees aan die Regulasies afgekondig by Provinsiale Kennisgewing nr. 553 van 1953.

Aansoeke moet gerig word aan die Mediese Superintendent, Dr. Van Niekerk Hospitaal, Springbok, en moet sy kantoor bereik nie later as 31 Desember 1956.

C. J. N. Basson
Mediese Superintendent

BELANGRIKE KENNISGEWING

Die Federale Raad verlang dat alle geneeshere, wat aansoek wil doen om kontrakpraktyk-aanstellings wat in hierdie kolomme geadverteer word, eers die Eresekretaris van dié Tak van die Mediese Vereniging van Suid-Afrika, binne wie se gebied die aanstelling val, moet raadpleeg sodat hulle kan weet of die voorwaardes van die aanstelling bevredigend is of nie. Kontrakpraktyk-aanstellings sluit in alle aanstellings deur Mediese Bystandsverenigings, Siekefondse en Fabrieke.

Die adresse van die Eresekretarisse van Takke is soos volg:

Grens: Dr. L. L. Alexander, Posbus 335, Oos-Londen.
Oos-Kaapland: Dr. Ella Britten, Hoogstraat 133, Grahamstad.
Midde-Kaapland: Dr. P. Jabkovitz, Glenairlie 1, Kaapweg 69, Port Elizabeth.
Wes-Kaapland: Dr. R. F. Maggs, Posbus 643, Kaapstad.
Oos-Rand: Dr. W. M. Bezuidenhout, Posbus 536, Benoni.
Griekwaland-Wes: Mnr. A. B. de Villiers Minnaar, Dutoitspanweg, Kimberley.
Natalse Kus: Dr. K. W. Dyer, Mediese Sentrum 112, Fieldstraat, Durban.
Natalse Binneland: Dr. T. H. Whitsitt, Posbus 285, Pietermartizburg.
Noord-Transvaal: Dr. E. Fasser, Administrasie-Gebou 28, Algemene Hospitaal, Pretoria.
Oranje-Vrystaat en Basoetoland: Dr. C. V. van der Merwe, Posbus 834, Bloemfontein.
Suid-Transvaal: Dr. W. Girdwood, Posbus 10102, Johannesburg.
Suidwes-Afrika: Dr. H. C. Paradisgarten, Posbus 1667, Windhoek.
Transkei: Dr. J. H. Hofmeyr, Posbus 318, Umtata.

Durban Blood Transfusion Service

MEDICAL DIRECTOR
PATHOLOGIST
PART-TIME MEDICAL OFFICER

Applications are invited from registered medical practitioners for the above appointments.

MEDICAL DIRECTOR

The inclusive emoluments of this post are £2,640 per annum and the post is not pensionable. The holder of this post will be responsible to the Committee of the Durban Blood Transfusion Service for overall control of the Service. He should have a practical experience in the running of a blood grouping and blood processing laboratory together with administrative experience in blood bank organization. It is desirable that the incumbent be a registered specialist.

PATHOLOGIST

The inclusive emoluments of this post will be of the order of £2,000 per annum, depending on experience, and the post is not pensionable. Applicants should preferably be registered specialists in pathology and the duties will consist of the laboratory control of the Blood Transfusion Service.

PART-TIME MEDICAL OFFICER

The emoluments of this post will be up to £1,800 per annum, dependent on experience and the proportion of time devoted to the Service. The duties consist primarily of the medical examination and bleeding of donors, both in Durban and with the Service's mobile unit.

Applications giving full details of experience and qualifications should be addressed to the Chairman of the Durban Blood Transfusion Service, P.O. Box 2324, Durban, on or before 15 February 1957.

Siekefonds van die Suid-Afrikaanse Spoorweë en Hawens

Aansoeke word ingewag van geregistreerde mediese praktisyns vir aanstelling in die volgende betrekking:

Spoorwegdokter vir die saamgestelde geneeskundige gebied, Pretoria-Noord en Wolmer. Salaris £194 per jaar.

Die aanstelling sal wees volgens grondslag van beperkte keuse van dokter en die salaris sal kwartaalliks gewysig word met ingang 1 Januarie 1957 in ooreenstemming met die getal lede wat verkies om op die lys van die suksesvolle applikant geplaas te word en sal teen die skaal van 18s. 6d. per voordeeltrekker per jaar bereken word.

Volle besonderhede in verband met die aanstelling kan verkry word van, en aansoeke gerig word aan:

Die Distriksekretaris, Oos-Transvaalse Distriksiekefondsraad, Chequergebou, 6de verdieping, Queenstraat, Pretoria.

L.W.—(i) Die aanstelling word op 'n deeltydse grondslag gemaak en behalwe die betrokke salaris is die gelde en toelaes waarvoor die siekefondsregulasies voorsiening maak, ook betaalbaar.

(ii) Werwing deur of ten behoewe van enige applikant stel so 'n applikant bloot aan diskwalifikasie.

Sluitingsdatum vir aansoeke, 23 Januarie 1957.

P. J. Klem
Hoofsekretaris

Johannesburg
9 Desember 1956

Anglo American Corporation of South Africa, Limited

LOCUM TENENS

A Locum Tenens is required by the Daggafontein Mine Native Hospital, Springs, Transvaal.

The successful applicant will be required to assume duty as soon as possible in the New Year for a period of approximately five months. Free board and lodging will be provided by the Mine Hospital.

The salary attached to the post is £110 per month, inclusive. A car allowance will be paid if the successful applicant provides his own motor car.

Applications in writing only, giving full details of experience, qualifications, age and marital status should be addressed to:

The Personnel Officer

P.O. Box 4587
Johannesburg

Stadsraad Pretoria-Noord

VAKATURE : DEELTYDSE MEDIESE GESONDHEIDSBEAMPTE

Aansoeke word hiermee ingewag vir 'n Deeltydse Mediese Gesondheidsbeampte, op die salarisskaal £120 per jaar (lewenskostetoelae ingesluit).

Van die suksesvolle applikant sal verwag word om 'n kontrak met die Stadsraad aan te gaan, soos voorgeskryf deur die Departement van Volksgesondheid.

Aansoeke moet melding maak van kwalifikasies, vorige ondervinding, huwelikstaat en ouderdom, en moet die ondergetekende bereik nie later as 12 uur Dinsdag, 8 Januarie 1957.

G. F. du Preez
Wnde. *Stadsklerk*

Vader Kestellpark
Posbus 52
Pretoria-Noord
11 Desember 1956
Kennisgewing nr. 43 van 1956

Vakante Deeltydse Distriksgeneesheerskappe

Aansoeke om ondergenoemde poste van distriksgeneeshere met vermelding van land van geboorte, kwalifikasies, ondervinding, vorige en teenswoordige betrekkings en die vroegste datum waarop diens aanvaar kan word, indien aangestel, word deur die Sekretaris van Gesondheid, Posbus 386, Pretoria, ingewag en moet hom voor of op 9 Januarie 1957, bereik. Afskrifte van getuigskrifte mag gestuur word.

Invloedwerwing deur of ten behoewe van 'n applikant stel hom bloot aan diskwalifikasie.

Die aanstelling is deeltyds en privaat praktyk word toegelaat.

Applikante moet ook vermeld of hulle albei amptelike tale kan praat, lees en skryf, asook of hulle melaatsheid en veneriese siekte kan diagnoseer.

Applikante moet verder vermeld of hulle ondervinding as 'n geneeskundige gesondheidsbeampte of in 'n soortgelyke hoedanigheid het. As om meer as een pos aansoek gedoen word, moet 'n afsonderlike aansoek ten opsigte van elkeen ingedien word.

Plek	*Salaris per jaar* £	*Toelaag vir medisyne per jaar* £
Transvaal		
Swartwater	625	25
Alldays	435	25
Kaapprovinsie		
Alicedale	100	20
Oranje Vrystaat		
Rouxville	350	65
Paul Roux	450	60

Die salaris dek alle gewone en roetine dienste, dog reistoelae teen 1/– per myl vir alle afstande wat buite 'n omtrek van drie myl vanaf die standplaas afgelê word, nagverblyf teen 15/– en bykomende vergoeding vir sekere dienste word betaal, asook gelde vir bywoning van hofsittings en geregtelike lykskouings ooreenkomstig die skaal van die Departement van Justisie.

Aansoekvorm en kopieë van kontrakvorms word op aansoek verskaf.

07948

UITSTEKENDE PRAKTYK TE KOOP
NOORD-KAAPLAND

'n Uitstekende en ongeopponeerde uitdeelpraktyk te verkry in 'n aangenaame Hospitaal dorp, langs die Vaal Rivier met onderwys en sport aangeleenthede. Chirurgie en kraam word gedoen. 'n Vennootskap praktyk geskik vir twee of drie Afrikaanssprekende praktisyns. Die netto inkomste per jaar van aanstellings allenig is voldoende om 'n lewe vir een vennoot te verskaf. Spreekkamers en twee woonhuise beskikbaar. Moet gesien word om dit te waardeer. Volle besonderhede op aanvraag. Skryf aan A.E.O., Posbus 643, Kaapstad.

Vacant Part-time District Surgeoncies

Applications for the undermentioned district surgeoncies accompanied by full particulars as to date and country of birth, qualifications, experience previous and present appointments of the applicants and the earliest date on which they can assume duty if appointed, should reach the Secretary for Health, P.O. Box 386, Pretoria, not later than 9 January 1957. Copies of testimonials may be submitted.

Canvassing by or on behalf of any applicant is liable to disqualify him.

The appointments are on a part-time basis and private practice is not precluded.

Applicants should state whether they can speak, read and write both official languages, also whether they are competent to diagnose leprosy and venereal diseases.

Applicants should also state whether they have any experience as a Medical Officer of Health or in any similar capacity. If more than one post is applied for, a separate application should be submitted in respect of each.

Place	*Salary per annum* £	*Drug allowance per annum* £
Transvaal		
Swartwater	625	25
Alldays	435	25
Cape Province		
Alicedale	100	20
Orange Free State		
Rouxville	350	65
Paul Roux	450	60

The salaries cover all ordinary and routine services but travelling allowances of 1/– per mile for all mileage travelled outside a radius of three miles from headquarters, night detention at 15/– and supplementary fees for certain other services will be payable, also fees for attention at courts and inquests in accordance with the tariff of the Department of Justice.

Forms of application and copies of draft agreement will be furnished on application.

07948

Departement van Gesondheid

VAKATURE VIR BESOEKENDE MEDIESE BEAMPTE (DEELTYDS)—KING GEORGE V-HOSPITAAL, DURBAN

Aansoeke om aanstelling in ondergenoemde betrekking in die personeel van die King George V-hospitaal, Durban, word van behoorlik gekwalifiseerde kandidate ingewag:

Betrekking: Neuropsigiater (Deeltyds). Honorarium aan Betrekking verbonde: £250 per jaar (vas).

Kandidate moet Suid-Afrikaanse burgers of burgers van 'n Statebondsland of die Republiek Ierland en tweetalig wees en moet minstens drie jaar in die Unie van Suid-Afrika of Suidwes-Afrika gewoon het.

Registrasie by die Suid-Afrikaanse Mediese en Tandheelkundige Raad as 'n neuropsigiater is 'n noodsaaklike vereiste vir aanstelling in die betrekking.

Die pligte sal raadgewend van aard wees en sal bestaan uit 'n gereelde weeklikse besoek om neurologiese en psigiatriese gevalle te sien en ook om noodgevalle van bogenoemde aard te behandel. Die hospitaal het 'n groot aantal gevalle van tuberkuleuse meningitis en daar sal van die aangestelde persoon verwag word om saam te werk in alle navorsing in verband met die onderwerp en om, waar moontlik, personeelsamespreking by te woon. Hy sal vir sy eie vervoer verantwoordelik wees en die koste verbonde daaraan, self moet dra.

Nadere besonderhede in verband met hierdie voorgenome aanstelling is by die Mediese Superintendent van die betrokke hospitaal verkrygbaar.

Daar moet aansoek gedoen word op die voorgeskrewe vorms (Z. 83 en Sdk. 8) wat van die Sekretaris van Gesondheid, Posbus 386, Pretoria, verkrygbaar is.

Die sluitingsdatum vir die ontvangs van aansoeke is 19 Januarie 1957.

07953

Provinsiale Administrasie van die Kaap die Goeie Hoop

HOSPITAALDEPARTEMENT

HOSPITAALDIENS : VAKATURES

1. Aansoeke word ingewag om die volgende vakante poste:

Pos	*Hospitaal*	*Emolumente*	*Sluitingsdatum*
Geneesheer, Graad B	Somerset-hospitaal, Groenpunt	£876×48—1,020 ×60—1,200 p.j.	9.1.57
Aansoeke moet aan die Mediese Superintendent gerig word.			
Geneesheer, Graad C	Livingstone-hospitaal, Port Elizabeth	£1,380 p.j.	19.1.57
(Departement van Kindersiektes—ongeveer 60 beddens.)			
Geneesheer, Graad C	Livingstone-hospitaal, Port Elizabeth	£1,380 p.j.	19.1.57

(Departement van Interne Geneeskunde.)

Die Livingstone-hospitaal, Port Elizabeth, is deur die Suid-Afrikaanse Mediese en Tandheelkundige Raad erken vir die doel van die reëls van die registrasie van spesialiste in die genoemde Departemente.

Aansoeke moet aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, gerig word.

2. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

3. Benewens die salarisskaal soos aangedui is 'n nie-pensioendraende lewenskostetoelae van £234 per jaar betaalbaar aan 'n voltydse getroude manlike beampte of werknemer.

4. Suksesvolle kandidate vir permanente aanstelling, indien nie reeds werksaam in die Hospitaaldiens nie, moet bevredigende geboorte-, gesondheids-, professionele- en registrasiesertifikate indien.

5. Kandidate word versoek om besonderhede aangaande die volgende te verstrek:

(*a*) Akademiese kwalifikasies (grade en diplomas verwerf en die standaard behaal in professionele eksamens; studiebeurse en spesiale toekennings).

(*b*) Professionele ondervinding (nie net 'n verklaring van die werkgewer nie maar ook besonderhede oor die inrigting waar die kandidaat gewerk het, en die tipe werk wat hy uitgevoer het).

(*c*) Name van drie persone na wie verwys kan word (van wie een verkieslik iemand moet wees wat werksaam is in dieselfde afdeling van geneeskunde as die kandidaat).

6. Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

7. Kandidate moet die vroegste datum waarop hulle diens kan aanvaar, meld.

M 464493

GLEN GREY MISSION HOSPITAL

Applications are invited for a vacant post at the abovementioned hospital. The successful applicant is required to do general medical duties for in- and out-patients. Surgical experience is essential. The hospital comprises 300 beds and a first class training centre for general and midwifery training. The applicant is also required to assist in lecturing. Salary scale as in Provincial Hospitals.

The Secretary

Glen Grey Mission Hospital
P.B. Queenstown, C.P.

FOR SALE

One Ultra-Sonic Machine, very little used. For surface and deep therapy. Mounted on stand with rubber wheels. Equipped with built-in stop-watch. Price as new £450 will sell for £250 or nearest offer. Write A.E.X., P.O. Box 643, Cape Town.

Provincial Administration of the Cape of Good Hope

UNIVERSITY OF CAPE TOWN : JOINT MEDICAL STAFF FOR GROOTE SCHUUR AND OTHER TEACHING HOSPITALS : VACANCY

Applications are invited from registered Medical Practitioners (registered Specialists) for appointment to the following post:

DEPARTMENT OF NEUROLOGY AND PSYCHIATRY

Medical Practitioner, Grade F (Psychiatrist), with salary at the rate of £1,980 per annum (fixed).

Initially the successful applicant will be offered a contract appointment up to 31 December 1957.

In addition to the basic salary, a non-pensionable cost of living allowance at rates prescribed from time to time by the Administrator (and at present amounting to £234 per annum), is payable to certain whole-time married officials and employees.

The conditions of service are prescribed in terms of the Hospital Service Ordinance, No. 19 of 1941, as amended, and the regulations framed thereunder, as well as the agreement entered into between the Provincial Administration and the University of Cape Town.

The Joint Medical Staff is required to serve jointly the Provincial Administration and the University of Cape Town.

Candidates must be registered Specialists in Psychiatry or must be in possession of an appropriate post-graduate diploma or degree, and are requested to furnish particulars in regard to the following:

(*a*) Academic achievements (degrees and diplomas held and the standard of achievement in professional examinations, scholarships and special awards).

(*b*) Professional experience (not only a statement of employer but the institution in which the individual worked and the type of work undertaken).

(*c*) Names of three referees (one of these should preferably be someone occupied in the same branch of medicine as the candidate), or to approach their referees to submit confidential reports to the Medical Superintendent before the closing date for the receipt of applications.

Application must be made in duplicate on the prescribed form Staff 23, which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Medical Superintendent of any Provincial Hospital, or the Secretary of any School Board Office in the Cape Province.

Applications must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 9 February 1957.

M 464492

Vacancy for Part-time General Medical Practitioner

DEPARTMENT OF DEFENCE

The abovementioned vacancy exists in Pretoria. The requirements are 19 hours per week which must be devoted to duties of the Department against a yearly remuneration of £762.

The successful applicant will do two clinics from 8 a.m. daily (excluding Sundays and Public Holidays) till all cases have been treated.

Applicants must be bilingual nationals of the Union of South Africa, and not older than 60 years, in possession of a recognized medical degree and must be registered with the SA Medical and Dental Council as a medical practitioner.

Applications for appointment must reach the Office of the Surgeon General, Defence Headquarters, Potgieter Street, Pretoria not later than 10 January 1957.

07887

Printed by National Commercial Printers, Elsies River, and Published by the Proprietors, *The Medical Association of South Africa*, Medical House, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'.

Vacancies for Medical Officers in the Public Service

1. Medical Inspector of Hospitals, £1,740 per annum (fixed).
Requirements: Registration as a medical practitioner with the South African Medical and Dental Council and experience in hospital administration.
Applications to the Provincial Secretary, Cape Town.
2. (*a*) Senior Medical Officer (Tuberculosis), £1,620 per annum (fixed), Windhoek.
(*b*) Regional Medical Officer, £1,500 per annum (fixed), Keetmanshoop and Grootfontein.
(*c*) Medical Inspector of Schools, £1,080×60—1,620.
(*d*) Medical Officer, Grade III or Grade II, £1,080×60—1,260 and £1,380 per annum (fixed) respectively, State Native Hospital, Windhoek.
Requirements: Registration as a medical practitioner with the South African Medical and Dental Council. Possession of a Diploma in Public Health will serve as a recommendation in the case of the posts of Regional Medical Officer. For appointment to the undermentioned posts, candidates must have had the following post registration medical experience:
Senior Medical Officer: At least five years. Experience in the combating of tuberculosis is essential.
Regional Medical Officer: At least three years.
Medical Officer, Grade II: At least two years.
Candidates appointed as Medical Officers, Grade III, will be considered for advancement to Medical Officer, Grade II, upon completion of two years medical experience after registration.
Applications to the Secretary for South West Africa, Windhoek.
3. (*a*) Medical Officer, Grade I, District Surgeon, £1,500 per annum (fixed), Bloemfontein, Durban, Kimberley, Knysna, Pietersburg and Potgietersrust.
(*b*) Medical Officer, Grade III or Grade II, District Surgeon, 1,080×60—1,260 and £1,380 per annum (fixed) respectively, Johannesburg, Kimberley, Louis Trichardt, Nylstroom, Pietersburg, Potgietersrus, Pretoria, Rustenburg, Vereeniging and Piet Retief.
(*c*) Medical Officer, Grade III or Grade II, Tuberculosis Service, £1,080×60—1,260 and £1,380 per annum (fixed) respectively, Durban, Restvale (C.P.), Johannesburg, Umtata, Kimberley and Cullinan (Transvaal).
(*d*) Medical Officer, Grade III or Grade II, Mental Hygiene Service, £1,080×60—1,260 and £1,380 per annum (fixed) respectively, various hospitals and institutions.
(*e*) Native Medical Officer, £828×48—972, Bulwer.
Requirements: Registration as a medical practitioner with the South African Medical and Dental Council. For appointment to the undermentioned posts, candidates must have had the following post registration medical experience.
Medical Officer, Grade I, District Surgeon: At least three years.
Medical Officer, Grade II and Medical Officer, Grade II, District Surgeon: At least two years.
Candidates appointed as Medical Officers, Grade III, will be considered for advancement to Medical Officer, Grade II, upon completion of two years medical experience after registration.
Applications to the Secretary for Health, Pretoria.
4. Medical Inspector of Schools, £1,080×60—1,620.
Requirements: Registration as a medical practitioner with the South African Medical and Dental Council.
Applications to the Provincial Secretary, Bloemfontein.
1. In addition to salary on the scales indicated, a cost of living allowance of £234 per annum is at present payable to married officers.
2. *Privileges:*
(*a*) Vacation Savings Bonus of 5% of basic salary, subject to certain conditions.
(*b*) Railway Travelling Concession once per year. (Approximately 40% discount on full tariff.)
(*c*) Membership of the Union and Widows Pension Funds.
(*d*) Payment of Subsistence and Transport expenses during absence from headquarters on official duty.
(*e*) Leave. Vacation: Up to 38 days per annum. Sick: 120 days with full pay and 120 days with half pay every three years.
(*f*) Official quarters are available for medical officers at most of the hospitals and institutions in the Union against payment of rent at 7½% of the valuation of quarters but not exceeding 12½% of salary and allowances. Personnel living in can obtain certain provisions through the local stores.
(*g*) Candidates appointed in South West Africa are paid a territorial allowance of £60 per annum if married and £30 per annum if single. In addition, an unfurnished house at nominal rent will be provided as soon as available.
3. Candidates must submit full particulars regarding qualifications and experience. Original certificates and testimonials must not be submitted.
4. Application must be made on forms Z. 83 and P.S.C. 8 (*a*) which are obtainable from the addresses indicated. Enquiries must also be directed to those addresses.
5. The closing date for the receipt of applications is 26 January 1957.

07976

Vakante Betrekking van Distriksgeneesheer

Aansoeke om die ondergenoemde pos van Distriksgeneesheer met vermelding van datum- en land van geboorte, kwalifikasies, ondervinding, vorige en teenswoordige betrekkings word deur die Sekretaris van Suidwes-Afrika, Windhoek, ingewag en moet hom nie later as 10 Januarie 1957, bereik nie.
Getuigskrifte (afskrifte) kan ingestuur word, maar geen versoek om ondersteuning van aansoeke word toegelaat nie. Applikante moet vermeld of hulle 'n kennis van albei amptelike tale besit. Die aanstelling is van 'n deeltydse aard en private praktyk word toegelaat.
Chirurgiese ervaring sal 'n aanbeveling wees. Applikante moet die vroegste datum waarop hulle diens kan aanvaar meld.

Distrik: Outjo.
Hoofkwartiere: Outjo, Suidwes-Afrika.
Salaris: £360 per jaar.

Die genoemde salaris dek alle gewone en roetine dienste maar reistoelae teen 1s. 6d. per myl vir alle afstande afgelê buite drie myl van die Hoofkwartiere, nagverblyf teen 22s. 6d. en bykomende vergoeding vir sekere ander dienste word betaal, en ook vergoeding vir bywoning van Hofsittings en ondersoeke, ooreenkomstig die tarief van die Afdeling Justisie van die Administrasie. Aansoeke moet ingedien word op vorm Z. 83, wat van enige Magistraatskantoor verkrygbaar is.

07969

Vacant District Surgeoncy

Applications for the undermentioned District Surgeoncy, accompanied by particulars as to date and country of birth, qualifications, experience and previous and present appointments of applicants, should reach the Secretary for South West Africa, Windhoek, not later than 10 January 1957.
Testimonials (copies) may be submitted, but canvassing by petition or otherwise should not be resorted to. The appointment is on a part-time basis and private practice is not precluded. Applicants should state whether they have a knowledge of both official languages. Surgical experience will be a recommendation. Applicants must state the earliest date on which they can assume duty.

District: Outjo.
Headquarters: Outjo, South West Africa.
Salary: £360 per annum.

The salary mentioned covers all ordinary and routine services, but travelling allowances at 1s. 6d. per mile for all mileage travelled beyond a radius of three miles from headquarters, night detention at 22s. 6d. and supplementary fees for certain other services will be payable; also fees for attendance at courts and inquests in accordance with the tariff of the Administration's Branch of Justice.
Applications should be submitted on form Z. 83 obtainable from any Magistrate's office.

07969